The Journal of the American Association of Nurse Anesthetists

IN THIS ISSUE

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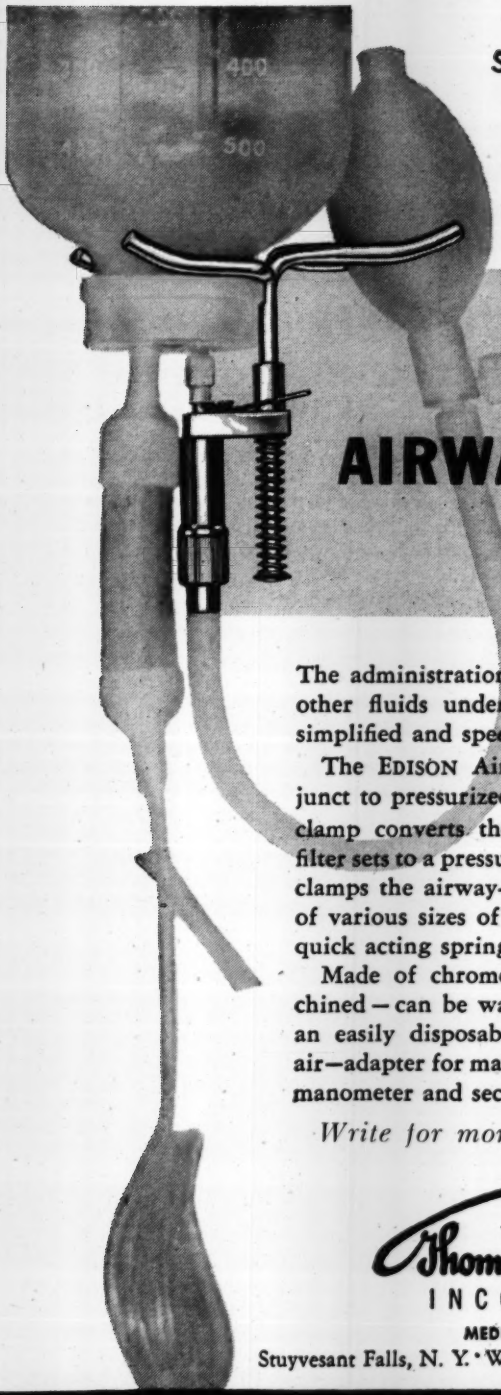

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Opinion Review

Is the Anesthetist Responsible for Teaching Others in the Hospital?

Inasmuch as we belong to a service occupation or profession, we owe safety to our patients. It was Florence Nightingale who said, The ". . . very first requirement in a hospital [is] that it should do the sick no harm." If we are vitally interested in safety, and it really is part of our job, then the total safety of the patient is of concern to us. And that includes all the time the patient is in the hospital (and afterward too) not only while we are anesthetizing him. Granting that this is so, then it is important that we teach others in the hospital all we can regarding anything that pertains to anesthetic or related emergency treatment.

How do you judge slight degrees of cyanosis? Tell those who come in contact with patients during the post-anesthesia period, in the emergency room and on medical services how to judge whether a patient is cyanotic and what it means to the safety of the patient's physical and mental health to correct this harmful state immediately. Show them what to do and how.

What constitutes full, adequate respiration? Tell those in charge of patients after anesthesia as well as those in the emergency room and on medical divisions what constitutes adequate respiratory exchange. This information may be life-saving, not only in the post-operative period but in the care of patients with severe asthma and other respiratory emergencies. If you were recovering from an anesthetic what skills would you like the person watching over you to have? Help teach those persons in your hospital the necessary skills.

If you entered the hospital as an accident victim, what would you like the person in the emergency room to be able to do for you? See that those persons responsible know how to do them.

What is it we are trying to do? Make every patient's life safer in every respect. That area which pertains to anesthetics and any related area is ours. We are supposed to know the answers. We can't possibly be available every second of every day in every room in the hospital. That is why we must teach others what to do in emergency situations.

Why did that patient die after a tonsillectomy before he recovered from the anesthetic? Could it have been because the nurse who was watching him didn't think it important to be with him every minute, or because she didn't recognize that mere movement of the patient's chest did not signify that he had a patent airway, because she didn't

know that gargling sounds should have been a signal to her to use a suction. Whose job is it to see that it never happens again? The anesthetist's. The anesthetist has a very special knowledge of respiratory problems. Having this knowledge, she is obligated to pass it on to others.

How should we do our teaching? Offer your help to the teaching staff of your hospital. Set up a class group. We are all in the business of patient care together. Keeping that in mind usually makes cooperation easy which in turn will make for safer patient care. One very effective method of getting information to others is in everyday contacts. When you put the patient back to bed, let those in attendance know not only what you want them to do but why. It only takes a few minutes. Surely the patient's life is worth that. Some day it may be your own life that is saved by your teaching.

Harriet Aberg, Galesburg, Illinois

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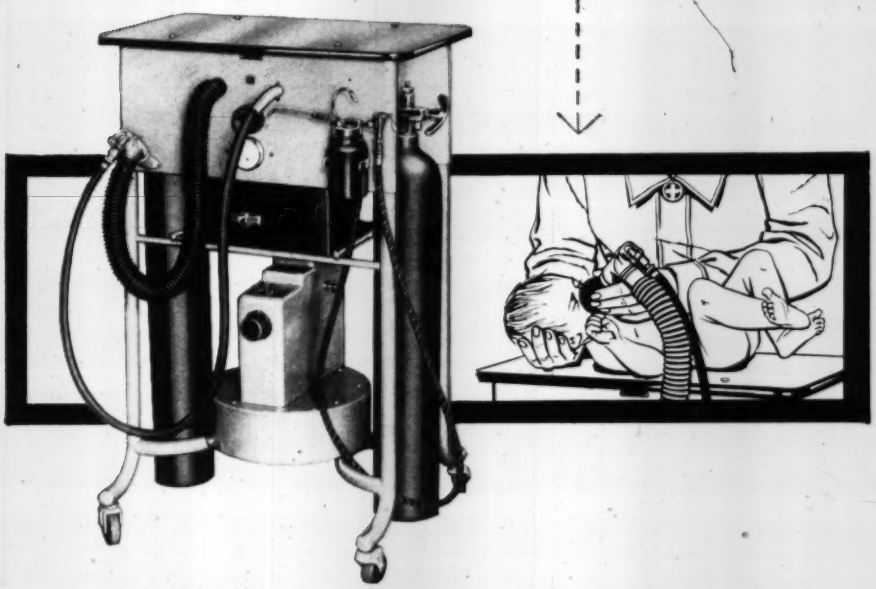

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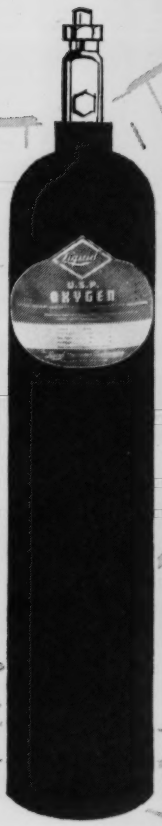

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The Journal OF THE
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Vol. XXII No. 1 February, 1954

Another Step Forward

The approved list of schools of anesthesia for nurses is published in this issue of the **Journal.** The list marks the completion of one phase of the "approval" program of this Association. However, in itself it has no meaning without a consideration of the development of the educational standards that have been the goal of this association since its organization in 1931. On January 19, 1952 a standard for the training of nurse anesthetists was adopted by the Board of Trustees following the recommendation of the Committee Advisory to the Approval Committee. Schools then in existence were given almost two years in which to raise the standards to the new requirements that had been outlined by the directors themselves. In a sense this association cannot say it raised the standards. More than half the schools had raised the standards to equal or exceed the minimums adopted by AANA at that time.

The great need for well-trained persons in the field of anesthesia has been the true stimulus for the other schools to improve the courses to meet the needs of surgeons for anesthesia adequate to the surgery being performed. Seventy-two schools of anesthesia have received certificates of full approval during two years since the new standards for approval were adopted. Thirty-two hospitals have opened schools of anesthesia for nurses.

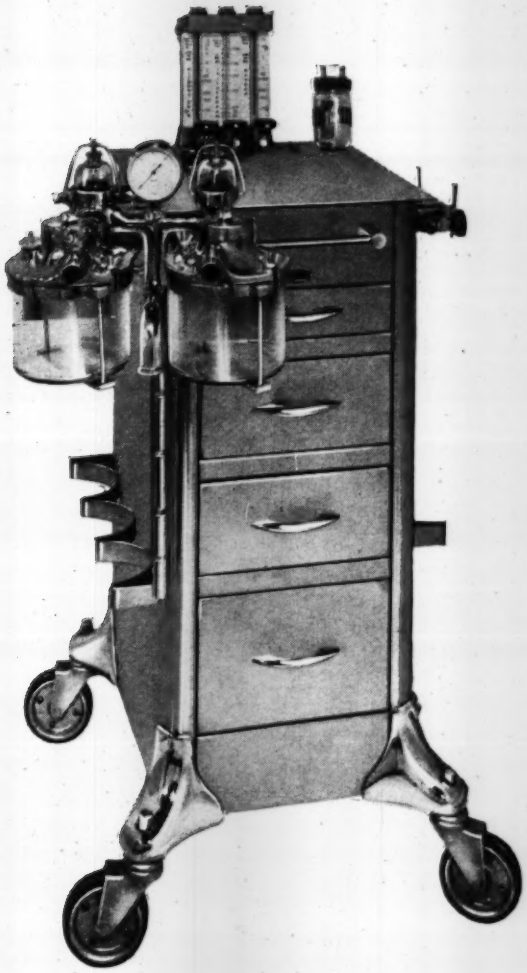
Unique in accreditation programs, the approval program for schools of anesthesia for nurses was financed by the members of the Association. Encouraged by the American Hospital Association Council on Professional Practices who in 1945 proposed that definite steps be taken to put the program of accreditation into operation and by the continued support of the American Hospital Association, the AANA has accomplished this major step in its program of education. Without the full cooperation of the members, the advisors, the directors, and the hospital administrators this program could not have reached this phase in so short a time. Schools of anesthesia have been **approved.** It is now the goal of the Association through continued help to the schools to continue to **improve** the schools.

Approved Schools of Anesthesia for Nurses

January 1954

This list includes all approved schools including those new schools that have been tentatively approved for one year on the basis of the standards adopted Jan. 19, 1952.

Jefferson-Hillman Hospital	Birmingham, Ala.
St. Vincent's Infirmary	Little Rock, Ark.
Letterman Army Hospital*	San Francisco, Calif.
Fitzsimons Army Hospital*	Denver, Colo.
St. Francis Hospital	Hartford, Conn.
Grace-New Haven Community Hospital	New Haven, Conn.
Hospital of St. Raphael	New Haven, Conn.
Walter Reed Army Hospital*	Washington, D. C.
Ravenswood Hospital	Chicago, Ill.
St. Mary of Nazareth Hospital	Chicago, Ill.
Wesley Memorial Hospital	Chicago, Ill.
St. Francis Hospital	Peoria, Ill.
St. John's Hospital	Springfield, Ill.
Ball Memorial Hospital	Muncie, Ind.
St. Joseph's Hospital	Lexington, Ky.
Charity Hospital	New Orleans, La.
Flint Goodridge Hospital	New Orleans, La.
Johns Hopkins Hospital	Baltimore, Md.
Boston City Hospital	Boston, Mass.
Massachusetts General Hospital	Boston, Mass.
St. John's Hospital	Lowell, Mass.
St. Luke's Hospital	Pittsfield, Mass.
Quincy City Hospital	Quincy, Mass.
Carney Hospital	Dorchester, Mass.
Mercy Hospital	Springfield, Mass.
St. Vincent's Hospital	Worcester, Mass.
University Hospital	Ann Arbor, Mich.
Grace Hospital	Detroit, Mich.
Mt. Carmel Mercy Hospital	Detroit, Mich.
Hurley Hospital	Flint, Mich.
St. Mary's Hospital	Duluth, Minn.
Minneapolis General Hospital	Minneapolis, Minn.
Mt. Sinai Hospital	Minneapolis, Minn.



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St. Joseph's Hospital	St. Paul, Minn.
Kansas City General Hospital	Kansas City, Mo.
Barnes Hospital	St. Louis, Mo.
St. John's Hospital	Springfield, Mo.
Creighton Memorial Hospital	Omaha, Nebr.
Albany Hospital	Albany, N. Y.
Kings County Hospital	Brooklyn, N. Y.
Long Island College Hospital	Brooklyn, N. Y.
Sampson AFB Hospital*	Geneva, N. Y.
Mary Immaculate Hospital	Jamaica, N. Y.
Queens General Hospital	Jamaica, N. Y.
Harlem Hospital	New York, N. Y.
New York Hospital	New York, N. Y.
St. Mary's Hospital	Rochester, N. Y.
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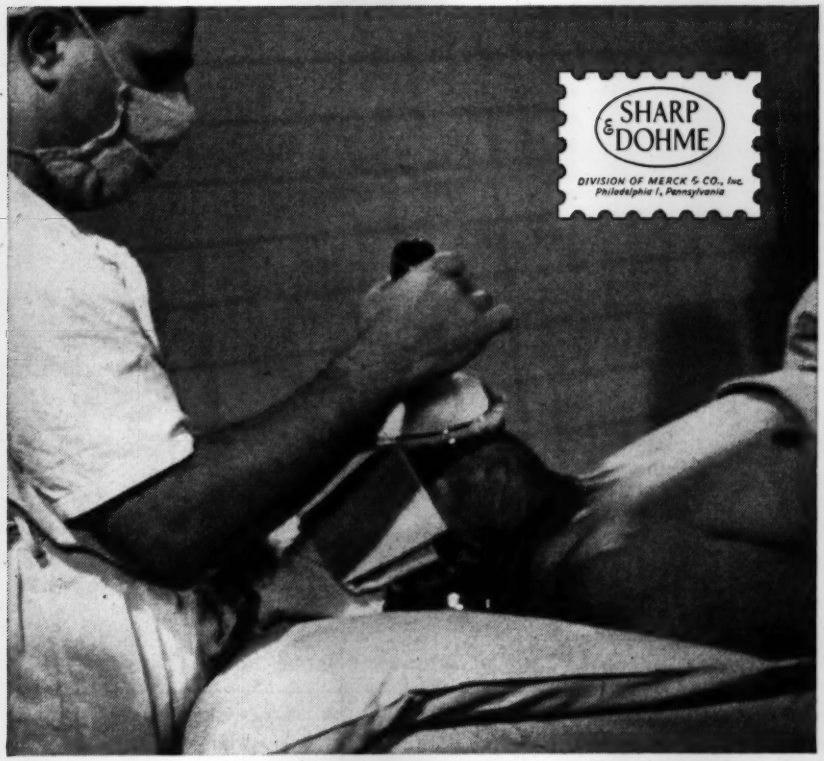
Duke University Hospital	Durham, N. C.
Watts Hospital	Durham, N. C.
North Carolina Baptist Hospital	Winston-Salem, N. C.
Cincinnati General Hospital	Cincinnati, Ohio
Ohio State University Hospital	Columbus, Ohio
St. Elizabeth's Hospital	Youngstown, Ohio
St. Vincent's Hospital	Portland, Ore.
Allentown General Hospital	Allentown, Pa.
Sacred Heart Hospital	Allentown, Pa.
St. Luke's Hospital	Bethlehem, Pa.
Thomas M. Fitzgerald Mercy Hospital	Darby, Pa.
Harrisburg Hospital	Harrisburg, Pa.
Lancaster General Hospital	Lancaster, Pa.
St. Joseph's Hospital	Lancaster, Pa.
Montgomery Hospital	Norristown, Pa.
Graduate Hospital	Philadelphia, Pa.
Jefferson Medical College Hospital	Philadelphia, Pa.
Pennsylvania Hospital	Philadelphia, Pa.
St. Agnes Hospital	Philadelphia, Pa.
Temple University Hospital	Philadelphia, Pa.
Woman's Hospital	Philadelphia, Pa.
Mercy Hospital	Pittsburgh, Pa.
Montefiore Hospital	Pittsburgh, Pa.
Presbyterian Hospital	Pittsburgh, Pa.
St. Francis Hospital	Pittsburgh, Pa.
St. John's General Hospital	Pittsburgh, Pa.



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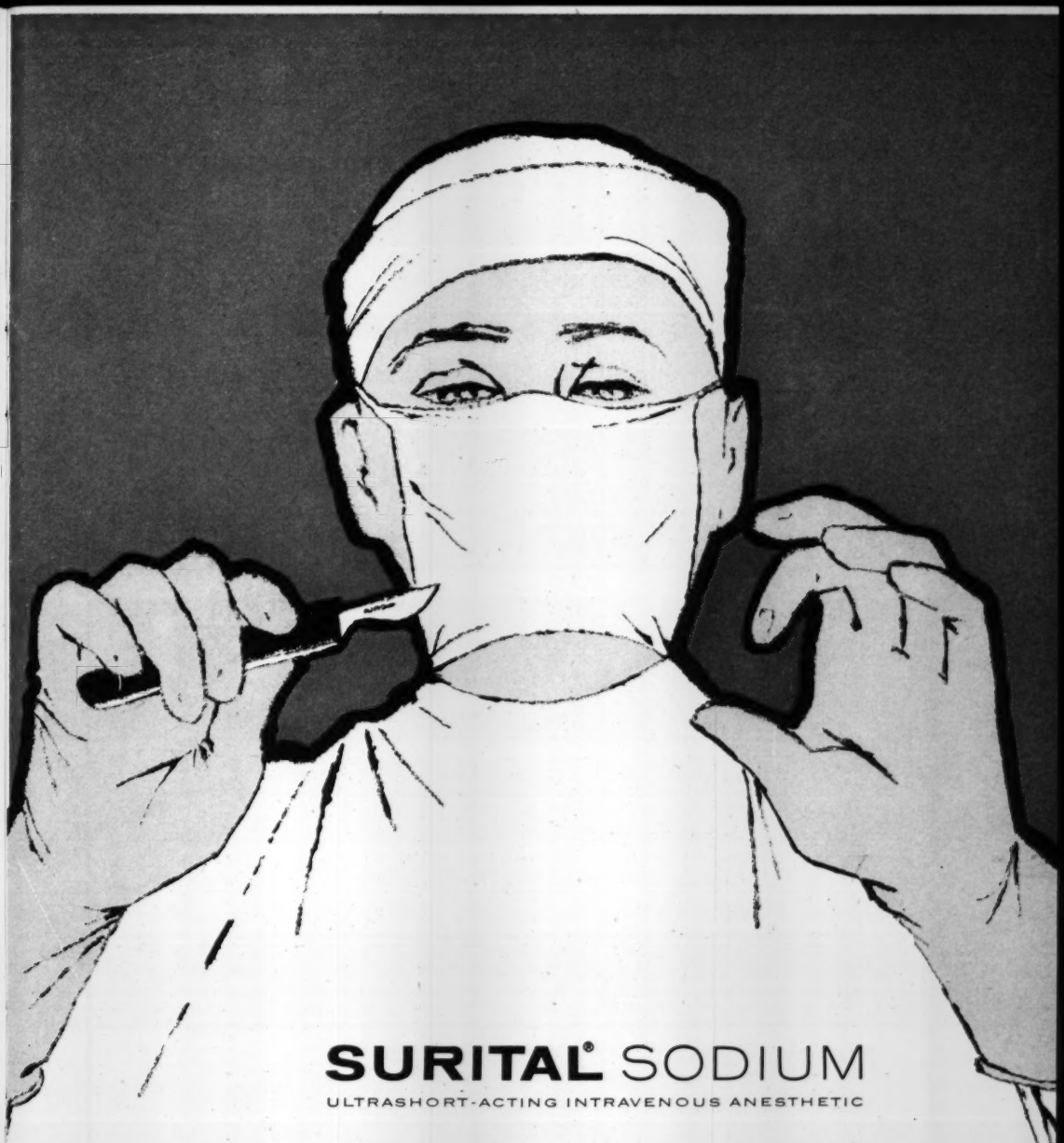
St. Luke's Hospital	Aberdeen, S. Dak.
St. John's Hospital	Huron, S. Dak.
Sacred Heart Hospital	Yankton, S. Dak.
Madison Sanitarium & Hospital	Madison College, Tenn.
University of Tennessee	Memphis, Tenn.
Oak Ridge Hospital	Oak Ridge, Tenn.
Baylor University Hospital	Dallas 1, Tex.
Brooke Army Hospital*	Ft. Sam Houston, Tex.
Harris Memorial Methodist Hospital	Ft. Worth, Tex.
Lackland AFB Hospital*	San Antonio, Tex.
De Paul Hospital	Norfolk, Va.
Norfolk General Hospital	Norfolk, Va.
Sacred Heart Hospital	Spokane, Wash.
Fairmont General Hospital	Fairmont, W. Va.
Luther Hospital	Eau Claire, Wis.
St. Francis Hospital	La Crosse, Wis.
St. Mary's Hospital	Wausau, Wis.

*Applicants accepted only from the respective branches of the government services.



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Inhalation Therapy

Mary Karp, M. D.*
Chicago

Inhalation therapy embraces a larger scope than oxygen administration alone. The huge alveolar bed presents an excellent absorptive surface, 500 times as large as that of the external body surface. The lungs are estimated to contain 300,000,000 alveoli, with a total surface of 65 square meters, which are lined with capillaries whose walls are only 0.000,0008 cm. thick. Inhaled gases are thus absorbed quickly into the blood stream and nebulized drug particles are carried to the most distal bronchioles and air sacs, causing thick mucosal and submucosal concentrations of the drugs as well as appreciable amounts in the circulating blood.

It is the anesthetist's fate to be inseparably wedded, thruout her professional life, to inhalation therapy. She must understand the methods of oxygen administration and in addition must be acquainted with advances in other phases of inhalation therapy. For the green oxygen tank is the emblem of the anesthetist and wherever it is included as a part of equipment is it assumed that the anesthetist will take charge, set up the equipment and give the treatment.

Read before the Tri-State Assembly of Nurse Anesthetists, Chicago, May 6, 1953.
*From the Department of Anesthesia, Division of Surgery, Northwestern University Medical School, and Department of Anesthesiology Wesley Memorial Hospital.

This paper will discuss three phases of inhalation therapy. It will introduce a method of oxygen administration which appears to give more comfort to the patient while maintaining a therapeutic concentration of alveolar oxygen; then will follow a short discussion on the use of alcohol vapor in the treatment of pulmonary edema and, finally, some remarks on the use of trypsin for liquefying tenacious secretions within the tracheo-bronchial tree.

The Method of Giving Oxygen Thru a Nostril Insert

Oxygen may be administered in many ways—by catheter, mask or tent. The most popular method at the present time utilizes the naso-pharyngeal catheter. Correctly inserted, catheters give therapeutic concentrations of 35-50% to the alveoli when the oxygen flows are maintained at 5-9 liters per minute from the oxygen tank.

The disadvantages of the nasal catheter are familiar to those acquainted with its clinical use. A tube such as the nasal catheter in the nasopharynx or oropharynx or both is uncomfortable and poorly tolerated, if at all, by some patients. Also, there is added discomfort from the withdrawal and insertion of catheters which must be changed frequently. An artificial humidifying mechanism must

be very efficient to keep the nasopharynx moist and uncrusted. The commercially available humidifiers which are attached to the regulators are incapable of saturating the oxygen with water particles and dry nasal membranes result.

Mindful of the many discomforts and disadvantages of present popular methods of oxygen administration, we have used a nostril insert device as an additional method for nasopharyngeal oxygen insufflation. Since 1945 we have had occasion to use it in over 8000 cases. As used in this hospital the nostril device consists of a blank end of French number 14 latex rubber catheter of the type usually used for oropharyngeal insufflation, plus a cylindrical piece of sponge rubber of a size to fit snugly into the external nares (fig. 1). The sponge discs may be cut conveniently in large numbers by means of a cork borer of suitable diameter, or may be prepared with ordinary bandage scissors. The device can be readily assembled with the aid of a hemostat which is pushed thru the center of the disc, and which then pulls the cut end of the catheter thru the disc, leaving it flush with the outer surface of the sponge. Because of the constricting tendencies of the sponge rubber, care must be taken that the lumen of the catheter is not interfered with, which might result in an annoying whistling sound. After the device is inserted into the external nares, the catheter is taped to the bridge of the nose and the forehead in the usual manner.

FIG. 1. Nostril Device for oxygen administration.

The remainder of the equipment employed is that usually available for oxygen administration; it consists of a large tank of oxygen, a regulator and a humidifier.

During the preliminary stages of our study with this method, it was important to discover the therapeutic significance of various oxygen flows. For this purpose a series of alveolar air samples was collected from anesthetized patients, first using the nasal catheter and then the nostril device, thus obtaining a comparative study.

All air samples were drawn endobronchially using a method similar to that employed by Rovenstine.[1] A leaded number 9 ureteral catheter was passed with the aid of a laryngoscope into a bronchus to a point about 6 cm. beyond the carina. The laryngoscope was withdrawn and the

1. Rovenstine, E. A., Taylor, I. B. and Lemmer, K. E.: Oropharyngeal Insufflation of Oxygen: Gas tensions in the Bronchus. Anesth. & Analg. 15: 10-13 (Jan. Feb.) 1936.

distal end of the catheter passed up through the nasopharynx and the external nares. The point of exit was found to be less disturbing than through the mouth.

Following the insertion of the ureteral catheter, the patient was returned to bed and a roentgenogram of the thorax was taken to confirm the position of the catheter (fig. 2).

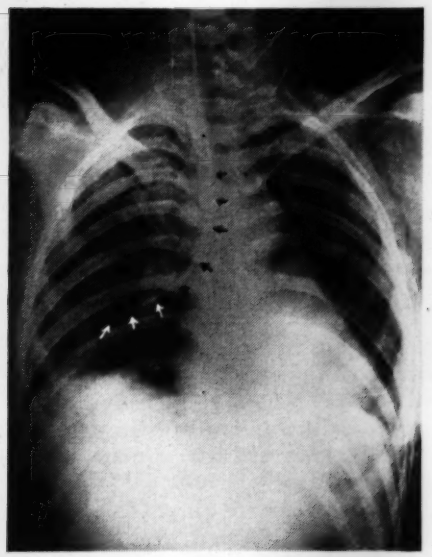

FIG. 2. Roentgenogram of Thorax Showing Ureteral Catheter in place.

Usually, patients were able to tolerate the catheter for several hours, and samples of exhaled air could be successfully withdrawn for analysis. A French Number 14 catheter was inserted into the oropharynx to a point opposite the tip of the uvula and then fastened securely to the forehead. Oxygen was started at a rate of 5 liters per minute and allowed to flow for thirty minutes, at the end of which time a sample was withdrawn. Immediately after sampling, the flow was increased to 7 liters per minute, maintained at this rate for thirty minutes and another sample withdrawn. After a 9 liter sample was collected the catheter was removed, the nostril insert installed and the procedure repeated.

The method of drawing samples was the same for all types of administration. To collect a sample, an airtight connection was established between the distal end of the ureteral catheter and the inlet of an evacuated gas sampling bulb. The stopcock of the bulb was opened near the end of each normal expiration and closed before the beginning of the next inspiration. This was done about three to six times or until the gas sampling bulb contained about 50 cc. of alveolar air at atmospheric pressure. The flow of oxygen was not interrupted during sampling.

All samples were analyzed for carbon dioxide and oxygen concentrations in the Van Slyke manometric gas apparatus, following the method of Van Slyke, Sendroy and Lin.[2]

Table 1 shows the results obtained with the first series of subjects. Since this was a comparative study, using the nasal catheter as standard, the result expressed as a percentage difference between the nostril device and the catheter method is thought to be valid. In the column titled "percentage difference" will be found the average amount by which alveolar oxygen concentrations obtained with the

2. Van Slyke, D. D.: Sendroy, J. Jr. and Liu, S. H.: Manometric Analysis of Gas Mixtures, III Manometric Determination of Carbon Dioxide Tension and pH of Blood: J. Biol. Chem. 95: 547-568 (March) 1932.

nasal catheter can be expected to exceed the alveolar oxygen concentrations obtained with the nostril insert method. We have found this difference to be 6.34 per cent for the 5 liter oxygen flow, 6.67 per cent for the 7 liter flow and 5.20 per cent for the 9 liter flow.

The advantages of the nostril device are derived primarily from the ease with which it is tolerated for long periods of continuous administration, a possible alveolar oxygen tension which compares favorably with that when the nasal catheter is used, and simplified maintenance. In our experience patients seldom complain of discomfort caused by the nostril insert device. This is in part due to the fact that oxygen

Table I.
Comparative Study
(Endobronchial Oxygen and Carbon Dioxide Determinations in volumes per cent)
of Nasal Catheter and Nostril insert.

	Liters per Minute					
Pt.	5		7		9	
	O_2	CO	O_2	CO_2	O_2	CO_2
	Nasal Catheter					
1	45.11	5.67	52.01	5.81	59.92	5.57
2	45.70	5.02	52.08	5.08	58.18	4.30
3	32.90	3.87	38.52	3.82	43.29	3.97
4	37.96	4.73	47.75	4.59	49.35	3.52
5	56.80	7.42	69.62	7.21	76.45	6.78
6	45.80	5.23	58.21	5.39	64.43	4.83
	Nostril Insert					
	37.79	4.37	41.31	5.25	46.90	3.33
	43.68	6.16	46.46	6.09	53.62	5.76
	30.54	4.88	32.21	4.71	40.64	4.80
	33.62	3.95	38.74	4.98	43.99	4.87
	49.15	6.91	63.16	7.17	75.14	7.17
	30.46	4.96	55.95	5.43	60.02	5.56

Table I, cont'd—Percentage of Difference

	5 Liters	7 Liters	9 Liters
	6.32	10.70	13.02
	2.02	5.62	4.56
	2.36	6.31	2.65
	4.34	9.01	5.36
	7.65	6.46	1.31
	15.34	2.26	4.41
Average % diff.	6.34	6.67	5.20

enters at the external nares which permits the utilization of a larger part of the natural humidifying mechanism than is possible with the nasal catheter, and in part owing to the absence of excess stimulation to the hairs of the nasal passages.

The ease of maintaining the nostril insert method of oxygen administration makes it especially advantageous clinically. The large sponge rubber sheets from which the insert devices are prepared are inexpensive and, as a result, they may be discarded after use. For greatest comfort we have found that a new device should be inserted once in each twelve hour period. However, no untoward complications will result should renewal be delayed for several hours or overlooked entirely.

The disadvantages of the nostril insert method are few and the difficulties presented sometimes are overcome by adjustment. The results in table 1 show that the nostril insert method of administration of oxygen offers a lower oxygen tension than is obtained with the nasal catheter. Since the two methods provide therapeutic alveolar concentrations, they would appear to be interchangeable in most instances of oxygen want. In cases in which higher oxygen concentrations are desired, the oxygen mask is the method of choice. Whenever cooling of the patient is needed the air conditioned tent is more suitable.

It is also true that a continuous stream of air flowing against the mucus membranes and turbinates may tend to irritate them. This difficulty usually is eliminated simply by shifting the insert from one nostril to the other, a task easily performed by the nurse or the patient when the necessity arises.

The most serious disadvantage of the nostril insert device is that nasal passages obstructed or occluded by mucus or deflected septa will not permit entry of oxygen. This difficulty can be dealt with by determining the patency of the nasal passages before the device is inserted into the nostril. Usually, nasal passages that will not permit oxygen therapy by means of the nostril insert

device will not permit oxygen therapy by nasal catheter, and face mask or tent must be used.

Alcohol Vapor in the Treatment of Pulmonary Edema

A fairly large amount of fluid may be tolerated in the lungs without seriously impairing the exchange of gases. However, in pulmonary edema there is transudation of blood elements thru altered alveolar capillary walls into lung air space, and this fluid is whipped into froth by respiratory activity. The bubbles interfere with the oxygen diffusion, and the conversion of the transudate to foam so increases its volume that gaseous exchange is progressively impeded. Anoxia ensues, followed by elevated pressure in the pulmonary artery and increased transudation. If this cycle is not broken, death from anoxia or suffocation occurs.

It is of prime importance to treat the underlying cause of the edema. This, however, may take time, and time may be sparing. Any means which will increase the airway will reduce the anoxia. If the foam could be eliminated from the fluid in the lungs, the airway would be cleared markedly. Since the stability of a foam depends on the character of the air-fluid interface—the surface tension of the fluid—any agent which would decrease the surface tension would make the foam disappear; would thus tend to break the vicious cycle and cause a reversible reaction.

In 1950 Luisada investigated several antifoaming agents in the treatment of pulmonary edema, using rabbits in which experimental acute pulmonary edema was induced by massive doses of epinephrine.[3]

Ethyl alcohol was found to be the most effective among the antifoaming agents, and it showed 2 separate effects; first, its antifoaming action which produces collapse of the foam bubbles; and, second, a general effect consisting of depression of the nerve centers, resulting in peripheral vaso-dilatation and decreased dyspnea. In his work on rabbits with pulmonary edema he was able to demonstrate that inhalations of alcohol vapor reduced foaming tendencies markedly. When morphine was given parenteraly in addition to the inhalation of alcohol, practically no edema was found in any of the rabbits.

Alcohol therapy was then tried on other types of experimental pulmonary edema, such as that induced by ingestion of ammonium chloride, and by rapid intra arterial infusion of isotonic saline solution. Results were similarly striking. Then normal subjects and cardiac patients were given alcohol inhalations in preliminary study. They tolerated the procedure well, and did not absorb sufficient alcohol thru mucosa of respiratory passages to produce anesthesia.

Luisada, satisfied with experimental work, used it clinically and reported gratifying results.[4] Inhalation of alcohol vapor promptly reduced foaming tendency; the quantity of alcohol absorbed was minimal and patients did not even

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3. Luisada, A. A.: Therapy of Paroxysmal Edema by Antifoaming agents: Circulation, 2:872 (December) 1950.

4. Luisada, A. A.: The Mechanism and Treatment of Pulmonary Edema: Medical Journal 100:252 (October) 1951.

become dizzy. At first he used this method when other methods failed. He now uses this treatment on all pulmonary edema cases.

The technic is simple. A mask-oxygen apparatus is used; 50% ethyl alcohol is placed in the humidifier bottle instead of water (Fig. 3). An an alternative, a nasal catheter and 95% alcohol may be employed; oxygen is begun gradually and within a few minutes a local anesthetic effect overcomes the irritation produced by these concentrations of alcohol. Given by mask the administration continues for 30 minutes; followed by 15 minutes of rest. Periods of administration and rest are then alternated until the patient shows signs of improvement. With the nasal catheter, administration is continuous until improvement is noted.

This procedure has definite advantages as an adjuvant therapy in its simplicity, its ready applicability to all types of pulmonary edema and its increasing use should bring about a substantial reduction in the mortality associated with this condition.

Trypsin Aerosol Therapy

Pure crystalline trypsin, extracted from the pancreas of all mammals, is a proteolytic enzyme. Tested in vitro, it rapidly dissolves fibrin clot and decreases the viscosity of empyema fluid. It will also digest dead tissue such as muscle, blood vessels, skin and fascia. It will not attack a living cell.

The anesthesiologist's interest in the use of crystalline trypsin lies in the ability of the product to "thin" and digest tenacious secretions within the tracheo bronchial tree.

The chief damage of retained secretions is airway obstruction. This may involve a lobule, segment, lobe or an entire lung and may result in atelectasis, emphysema or infection behind the ob-

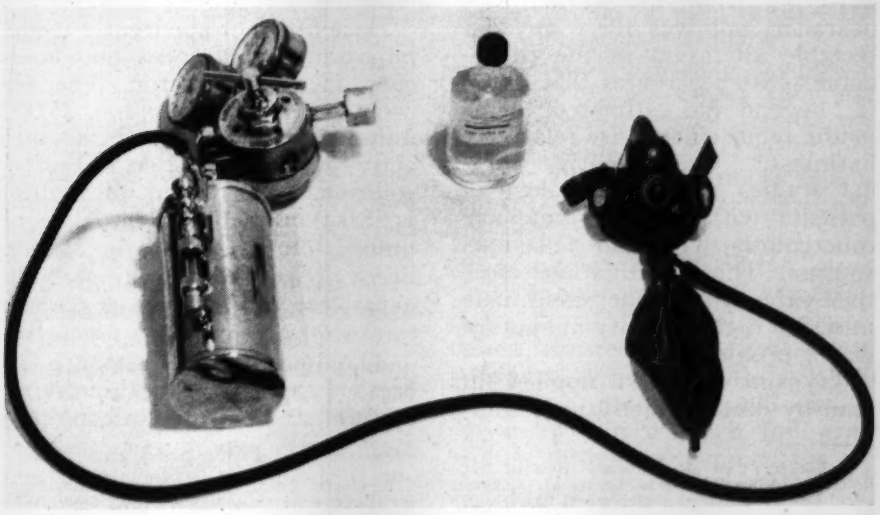

Fig. 3. Apparatus for administration of Alcohol Vapor.

struction. Some of these sequalae require prolonged medical care and surgical removal of affected tissues and may be a cause of disability or limited activity for long periods of time. Hence the importance of liquefying and removing secretions before the occurrence of airway obstruction and the potential complications.

The vigorous fibrinolytic properties of trypsin plus its ability to cause digestion of respiratory mucus indicate the value of the enzyme in the treatment of disorders of the bronchial tree associated with the hypersecretion and accumulation of fibrinomucinous material. Trypsin acts by effectively removing the clogging secretions.[5]

Experimentally, trypsin produces rapid liquefication of samples of thick sputum obtained from patients with active pulmonary tuberculosis. Microscopic examination of the tracheo bronchiol tree of rabbits following the use of aerosol trypsin showed no deviation from the normal appearance, nor was there any detectable alteration in the ciliary action. Studies reveal that doses far beyond the estimated therapeutic requirements are relatively harmless.[5] Consequently, clinical studies were undertaken in patients with active pulmonary tuberculosis who had tenacious sputum. The investigations were then extended to other conditions in which high viscosity of sputum was a problem.

Trypsin was first applied in man by directly instilling a solution into the bronchoscope. The most satisfactory method at present is direct application by means of a mask, nebulizer, rubber tubing, and a source of oxygen.

Trypsin is effective over a PH of 5 to 8, exhibiting its greatest activity at PH of 7.1. Trypsin has a broad spectrum of proteolytic action on proteins, denatured proteins, true peptones, mucin, fibrin and protein split products. The final degradation products of trypsin digestion are small polypeptides and some amino acids.

The enzyme is stable indefinitely in dry form at room temperature. The vehicle in which it is diluted is Sorenson's Phosphate buffer solution (prepared with pyrogen-free water and U. S. P. Salts); because 40-50% of the proteolytic activity is dissipated within three hours, the solution should be prepared freshly each time immediately prior to its use.

Method of administration is by means of a nebulizer, mask regulator and oxygen tank (Fig. 4).

Dose of agent apparently is not important; concentration however, is a vital factor. The accepted dose for a child is 25,000 units of trypsin (25 mgs. tryptic activity) diluted in 1 cc. of buffer-solution.[6] The initial adult dose is 75,000 units diluted into 3 cc. of buffer solution. Usually 5 to 6 liters of oxygen per minute will aerosolize 1/2 to 1 cc. of trypsin mixture in 5 minutes.

The immediate effects are increased expectoration, thinning of sputum, and increase in coughing

5. Linker, Carl R., Reiser, Howard G., Rolthig, L. Chandler, Curtis, George M.: Enzymatic Lysis of Respiratory Secretions by Aerosol Trypsin: J. A. M. A. Vol. 149, pp 816-821 (June 28) 1952.

6. Unger, Leon, M. D.: Trypsin Inhalations in Respiratory Conditions Associated With Thick Sputum: Accepted for Publication, J. A. M. A.

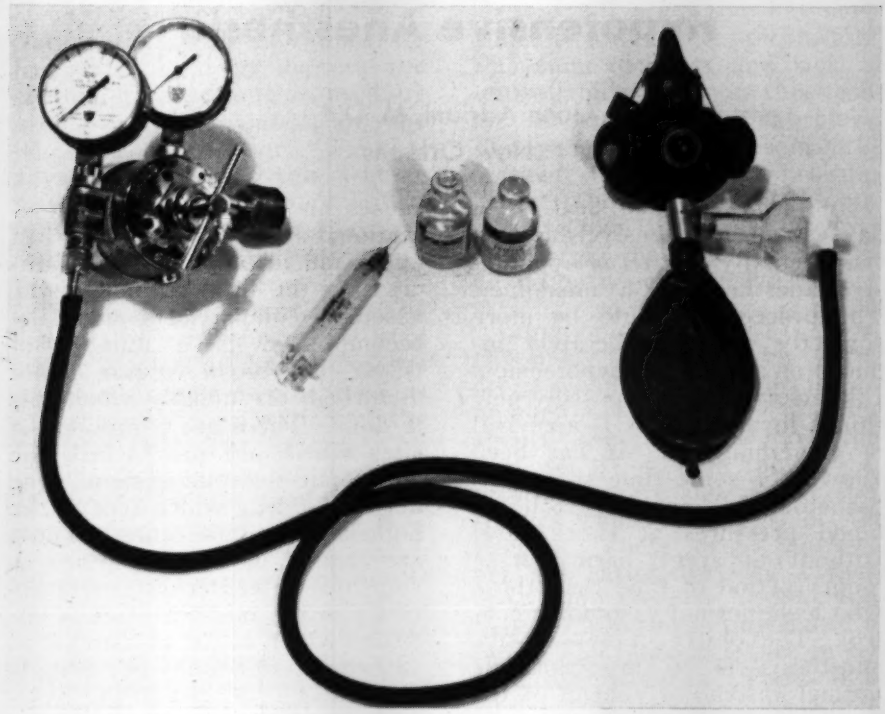

FIG. 4. Apparatus for Administration of Trypsin by inhalation.

due to stimulation of the cough reflex associated with liquefication of the bronchial secretions. This occurs immediately after and during inhalation, and usually terminates within three hours. Transient hoarseness has been noted if the oxygen flow is too high or droplet size too large, or if concentration of trypsin is more than 50,000 units cc.

Treatments may be given once or twice daily. Our routine is to give 75,000 units the first day, 100,000 the second day, and 125,000 the third day.[6] Following one week of rest, three more treatments may be given, if indicated. After the administration of trypsin, a small amount of water should be used to rinse the oral and nasal passages.

Side reactions occur in approximately 5% of cases; they are hoarseness, dyspnea, chills and fever. Chills and fever occur 6-12 hours after treatment. Dyspnea may be seen shortly after completion of treatment.

Administration of 50 mg. of an antihistamine and 10 gr. of acetylsalicylic acid will eliminate almost all reactions (hoarseness, dyspnea, hyperpyrexia).

Trypsin aerosol should not be given within a week following frank pulmonary hemorrhage.

Trypsin is indicated for the

(Continued on page 71)

Hypotensive Anesthesia

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One hears a good deal these days about hypotensive anesthesia. Actually the term hypotensive anesthesia is a misnomer. The procedure should be more correctly called deliberately induced or intentional hypotension. Like many other supposedly new things in medicine it is a revival of something old. It has been known for some time that sympathetomized subjects tolerate blood pressures at shock level without apparent harm for a longer period of time than those who have normal vasomotor control. The object in hypotensive anesthesia is to interrupt the normal vasomotor control of the vascular system and induce a hypotension. This hypotension is utilized at a time when massive hemorrhage is anticipated. The technic therefore is one that is adaptable to formidable types of surgery in which uncontrollable hemorrhage is a possibility. The hypotension reduces the amount of blood loss.

The autonomic nervous system consists of a plexus of nerves and ganglia, which supply the heart, blood vessels, glands and viscera. The vascular system is supplied by both vasoconstrictor impulses and vasodilator nerves. A blockade of vasoconstrictor impulses or an increase of dilator impulses causes an increase in the cross-sectional area of the vascular bed and a fall in blood pressure. Increasing the total volume of the vascular compartment may be accomplished in a number of ways. The blood volume is not disturbed, presumably when this is done. There are a number of sites where one may attack the autonomic nervous system. One may use a drug which acts on the highest autonomic centers. These are located in the mid-brain. A sympathetic center located anteriorly in the mid-brain sends impulses which cause an increase in vasoconstrictor tone. If a drug is used which depresses the sympathetic center vasodilatation occurs. Apresoline is an example of such a drug. The number and frequency of sympathetic impulses decreases and vasodilatation results. The use of this drug during surgical anesthesia is not practical however. The sympathetic centers and parasympathetic centers send fibers down to the medulla, to the vasomotor center. The vasomotor center has a vasodilator component and a vasoconstrictor component. One may thus cause vasodilatation by using drugs which affect the vasomotor center. The vasomotor center may be depressed so that the vasoconstrictor component sends fewer impulses to the blood vessels and relaxation occurs. Narcotic and hypnotic drugs accomplish this to a certain extent.

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Heavy doses of barbiturates in combination with morphine are frequently used to depress the center. This technic is used by some otolaryngologists in the fenestration operation. The disadvantage in using this technic is that the other centers in the medulla, notably the respiratory centers, are depressed along with the vasomotor center.

If a drug which stimulates the vasodilator component of the center is used, an increase in the impulses passing down the vagus nerve causes a relaxation of the vascular bed. The veratrum alkaloids behave in this manner. Likewise they are not of practical use during surgery because they lack controllability. The attack of the vasomotor control in the medulla then is of no practical significance from the standpoint of surgical anesthesia.

The pre-ganglionic fibers emerge from the spinal cord and pass to the sympathetic ganglia. The sympathetic ganglia then send post-ganglionic fibers to the blood vessels. One may attack and cause a blockade of the preganglionic fibers as they emerge from the spinal cord. The impulses then disappear and the blood vessels become relaxed. This may be accomplished by injecting procaine into subarachnoid space by the procedure known as total spinal block. This technic has been used to a large extent by the British. Its chief drawback is its lack of controllability. The blood pressure sometimes is completely imperceptible and instances of cardiac arrest have not been uncommon. Peridural injections act in the same manner as subarachnoid injections but are somewhat more difficult to execute technically. The same dangers and lack of controllability exist in this technic as with total spinal block. The ganglia themselves may be blocked by injecting procaine paravertebrally. This is somewhat tedious and is not effective in securing the generalized vasodilatation which is required.

Ganglionic blockade is more effectively obtained by using drugs which act systemically. A number of ganglionic blocking agents are available for this purpose. It is well known that nicotine, curare and similar substances cause paralysis of the ganglia. These drugs however are not satisfactory for clinical use. More recently hexamethonium or C_6 has been introduced to reduce the blood pressure in hypotensive patients. Tetra-ethyl-ammonium chloride (Etamon) has also been used for the purpose. Neither hexamethonium nor Etamon are entirely satisfactory for the purpose. Hexamethonium frequently induces a sustained hypotension which is not easily reversed. Etamon frequently has a fleeting action and the hypotension is not easily controlled. Two newer preparations which are being studied are Arfonad and Pendiomide. Both these drugs appear to be controllable and short-acting. They thus permit greater control of the pressure and prompt reversal of the vasodilatation when it is desired to interrupt it. The method of using ganglionic blocking agents is the most popular and the most effective way of inducing controlled hypotension.

If desired the vasodilatation may be obtained by causing a blockade at the nerve endings of the autonomic nervous system. A sympatholytic drug or adrenolitic drug may be used. Dibenamine, Priscol and Benodaine are examples of drugs which cause a blockade at the sympathetic nerve endings. Although these drugs are effective they are used less frequently than the ganglionic blocking agents for inducing hypotension during surgery.

One may go beyond the nerve endings and administer a drug which depresses the smooth muscle of the vascular bed directly. The nitrates and nitrites may be used for this purpose. However, they are not controllable or their actions are evanescent and for this reason hypotensive anesthesia is rarely induced with them.

The blood pressure may also be lowered by reducing the blood volume by employing the procedure known as arteriotomy. A canulla is placed in the proximal end of the divided radial artery at the wrist. The distal end is ligated. Blood is withdrawn temporarily into a flask containing an anti-coagulant, usually heparin or sodium citrate. A manometer is placed in the system so that the blood pressure can be read directly. As soon as the need for the hypotension is over the blood is re-transfused into the artery under pressure. Obviously in this method the vascular system retains its vasomotor control and the generalized vasodilatation which is desired in hypotensive anesthesia is not present. In due time irreversible shock may occur. Frequently when this technic is employed it has been noted that all the blood withdrawn cannot be replaced. Presumably this is due to the fact that the generalized vasoconstriction has occurred from the hemorrhage and that the vascular bed has shrunk in size. Arteriotomy is a tedious, cumbersome procedure and not easily managed. Certainly it is not as simple as injecting a drug intravenously as is done when the ganglionic blocking agents are used. It must be remembered also that it involves sacrifice of the radial artery.

The chief use of controlled hypotension is for situations in which considerable bleeding is anticipated. The hypotension is induced, at least ten minutes prior to the moment the bleeding is anticipated. If it is not reduced before this time it is nowhere near as effective as desired. A relatively bloodless field results for a brief period so that the surgery may be accomplished with greater ease and with a minimum of blood loss. Controlled hypotension facilitates neurosurgery, various forms of plastic surgery, surgery about the head and neck in which bleeding is anticipated and radical pelvic surgery. In the present light of our knowledge the technic is best reserved for patients who have malignant disease or benign conditions for which operation is a necessity but in which blood loss may cause a loss of life.

Obviously there are complications and hazards in using a method of this sort. It cannot be employed routinely for every case. The relaxation of the vascular bed reduces the blood flow through all vital organs. If the

cardio-vascular system is diseased the incidence of complications and untoward reactions and undesirable sequela mounts. Circulation time is prolonged sometimes twice the normal rate. If the coronary arteries are diseased the myocardium does not receive an adequate blood supply and coronary insufficiency follows. Some of the fatalities which have occurred during and after the procedure have been due to coronary thrombosis. The brain likewise may suffer damage due to the lack of nourishment or oxygen deficiency. The same pathologic findings one would find after a period of acute anoxia occur under these circumstances also. The slowing of the circulatory system in patients who have sclerotic vessels predisposes to thrombosis which may cause emboli to pass to the brain and other organs. The emboli however have been confined largely to the cerebral vessels. Cerebral thrombosis and coronary thrombosis appear to be the most frequent complications of the technic.

At least 60 mm. of mercury pressure is necessary to maintain adequate blood flow through the kidney. If the blood pressure remains below this level for any length of time filtration ceases and damage to the tubules may result. Anuria then follows in the post-operative period. The picture in these cases is similar to that noted in the lower nephron syndrome.

In executing the technic the patient is anesthetized in the usual manner preferably with ether or a drug which does not cause vasodilatation. Ten to fifteen minutes before the anticipated time of excessive bleeding the vasodilator drug is administered intravenously. Usually the blood pressure is maintained between 60 to 80 millimeters of mercury. If it is maintained above 80 millimeters of mercury the results are not satisfactory. Bleeding continues and is not curtailed. The technic controls only the ooze and not bleeding from a major vessel.

The blood in the vascular space is influenced by gravity after the blockade is induced and may be shifted about at will. The blood pressure may be reduced further by inclining the patient with the head up and feet down. If the pressure falls below the desired depth the head may be tilted downward and the feet raised. In this position the effects of gravity cause an increase in the amount of blood returned to the heart and brain and the blood pressure then rises. After the denervation is instituted the blood pressure is controlled by varying the position of the patient until the effects of the drug wear off. The dose then is repeated. It is advisable not to allow the blood pressure to fall below 60 millimeters of mercury. Blood must be replaced as lost otherwise irreversible shock may occur. These patients do not tolerate blood loss. It is noteworthy that little anesthetic is required during the period of hypotension. The depth of anesthesia necessary is nowhere near as great as that which is required without hypotension. The period of hypotension should be as brief as possible, not to exceed one hour as a rule. At the conclusion of the portion of the operation requiring the hypotension the

blood pressure may be raised by tilting the patient or by injecting a vasoconstrictor drug such as neosynepherin, arteronol or desoxyephedrine. In some cases a combination of tilting and vasopressor drug may be required to restore the blood pressure to normal.

The most frequent complication noted in the post-operative period is slow recovery. Patients require a long time to recover from the post-anesthetic depression. The second most frequent complication is reactionary hemorrhage. Bleeding points which have not been detected and tied off before closure may resume bleeding when the blood pressure rises. Anuria, cerebral thrombosis, apoplexy, coronary thrombosis, pulmonary embolus and various types of encephalopathy are occasional complications encountered. Decerebration from damage to the cerebral cortex due to impaired blood supply is not an uncommon occurrence. One cannot deny that the technic facilitates the more formidable types of surgery. If used with discretion in malignant conditions it is a worthwhile procedure notwithstanding the possible complications. It is not an anesthetic procedure by any means. The anesthetist merely executes the technic because he ordinarily watches the blood pressure for the surgeon and is in the position to direct its management. The procedure is actually one of disturbing the physiology during surgery in order to accomplish vasodilatation.

Anaesthesia Problems in Cardiac Surgery

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Anaesthesia for cardiovascular surgery has become a frequent requirement in many hospitals. The recent progress in the surgery of the heart and great vessels has been parallel to and dependent upon a greater understanding of cardiorespiratory physiology and anaesthesia.

The care of the patient who is to undergo cardiac surgery requires the closest cooperation between the cardiologist, the anaesthetist and the surgeon during the preoperative, operative and postoperative periods. The anaesthestist contributes special knowledge of cardiorespiratory physiology and anaesthesia, and should also have a clear concept of (1) the types of disease amenable to surgical intervention, (2) the methods of preoperative evaluation and care, (3) the factors concerned in the choice of anaesthesia and its management during surgery, and (4) the postoperative care of the cardiac patient.

In general the reason that surgery can be successfully applied to many congenital and acquired diseases of the heart is that the defects are of a **mechanical** nature and therefore subject to **mechanical** correction. Functionally, the heart may be considered as two separate pumps. The right heart pump receives blood from the great veins and pumps it into the capillary bed of the lung and the left heart pump receives blood from the pulmonary veins and pumps it into the peripheral arterial tree. The auricle, or atrium, of each heart acts as a sort of collecting chamber and funnel which passes blood through a valve into the chamber of the ventricle. The ventricle is the pumping chamber that propels blood out through a valve into a large artery. With each synchronous beat, each side of the heart puts out about equal quantities of blood. That is, the stroke volume output of the right side usually equals the stroke volume output of the left side. Any continued inequality of volume output between the right and left sides of the heart, either during rest or exercise, produces definite physiological abnormality. Abnormalities of direction of blood flow or anomalous openings between the chambers of the heart or between the great vessels is another cause of abnormal physiology. The anatomical defects which may occur are; in the great vessels, the valves, the myocardium, the nourishing ves-

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sels of the heart muscle, or the septa dividing the right and left sides of the heart. These conditions may be congenital or acquired, and may occur singly or in combination.

Examples of Heart Diseases Amenable to Surgical Correction

A clear concept on the anatomical and physiological problem presented by the defect which is to be operated upon, and the method of surgical correction is important to the anaesthetist since these considerations are essential in the anaesthetist's planning and handling of the case. It is therefore worthwhile to review some of these conditions and their method of surgical correction. The first two conditions are well known to you since their surgical treatment has been highly successful and they are the oldest cardiovascular procedures. [1,2]

The first is patent ductus arteriosis. Of course the open ductus arteriosis is a normal situation in the foetus. It normally closes in the first several weeks after birth. The effect of the non-closure is that a large amount of arterial blood from the aorta flows into the pulmonary artery and never reaches the peripheral circulation, but recirculates through the lung and returns to the left heart. The size of the patent ductus may vary. Some patients have lived a full life with this condition, but patients with patent ductus usually die before the age of forty. The surgical correction of this condition consists simply in the ligation or division of the patent vessel between the aorta and pulmonary artery. This is done with a mortality of less than 1% and restores the patient to an essentially normal life expectancy.

Coarctation of the aorta is a narrowing or stenosis of the aorta, usually just beyond the left subclavian artery. This narrow point is hardly large enough to permit the passage of a sucker stick. The result is that there is great increase in blood pressure in the vessels and branches proximal to the coarctation. The blood supplied to the body below the coarctation must come principally from collateral circulation, derived from the vessels leaving the aorta, proximal to the coarctation. The amount of blood supplied to the lower half of the body is often adequate only to maintain life and moderate activity. The upper half of the body has blood under too much pressure while the lower half of the body has blood under inadequate pressure. The heart constantly works against a pressure head which is very high. The patient usually succumbs in the early thirties because of heart failure, aortic rupture or cerebral vessel thrombosis or hemorrhage. The surgical correction of this condition is to excise the point of coarctation and to reanastomose the aorta allowing a normal flow of blood. This can be accomplished with a mortality rate of about 7% and if done before irreversible changes have occurred offers normal outlook for life and health.

Constrictive pericarditis is a

1. Gross, R. E., and Hubbard, J. P.: Surgical ligation of a patent ductus arteriosus; report of first successful case. J.A.M.A., 112:729, 1939.
2. Crafoord, C., and Nylin, G.: Congenital coarctation of the aorta and its surgical treatment. J. Thoracic Surg., 14:347, 1945.

condition in which the pericardium becomes scarred, thickened and contracted in such a way as to progressively strangle the heart and the great vessels. As the compression is greater and greater the heart is less and less able to dilate or relax in diastole and so the chambers cannot adequately fill, due to the increasing constriction of the tight pericardium. This condition is corrected by releasing the heart from its confinement by cutting away the scarred pericardium so that most of it is removed and there is adequate filling of the heart chambers in diastole with marked improvement in the circulation.

Within the heart, defective function of the valves may be subject to correction.[3] Occasionally a patient is born with a marked narrowing or stenosis of the pulmonary valve at the outflow point from the right ventricle. This may or may not be associated with other anomalies and produces an obstruction to the outflow of the right ventricle. If this is not corrected the life expectancy may be twenty years or less. The correction of the stenosis consists in cutting the diaphragm of the stenotic valve so that adequate flow is permitted. This is done by introducing a cutting instrument through the right ventricular wall and passing it up through the funnel produced by the stenotic valve and then cutting the stenotic valve diaphragm.

Rheumatic mitral stenosis has proven to be greatly benefited by surgical intervention.[4] This is caused by progressive adhesion of the two main cusps of the mitral valve so that the opening is progressively diminished with the result that at first insufficient quantity of blood can pass through the open valve to meet the requirements of exercise, and later the flow through the valve is inadequate to permit even moderate activity. This inequality in pumping capacity between the right and left hearts due to the mitral stenosis leads to pulmonary congestive failure, right heart crippling, peripheral congestive failure and eventual death. Surgically this condition is approached through the left auricular appendage. The stenosis of the mitral valve is opened by finger separation of the leaflettes or by cutting them apart along the lines of their adhesion with a small knife attached to the finger. Postoperatively the patient experiences tremendous increase in the capacity for activity.

Inadequate coronary artery flow due to coronary arteriosclerosis is a debilitating condition because the heart muscle itself receives inadequate amounts of blood which produces muscle ischemia during exercise or emotion referred to as angina pectoris, or the patient may have myocardial infarction. Dr. Claude Beck has labored mightily on this problem and it appears that a solution is at hand.[5] This is accomplished in two stages. At the first operation, a blood vessel is grafted

3. Brock, R. C.: Pulmonary valvulotomy for the relief of congenital pulmonic stenosis. Brit. M.J. 1:1121, 1948.

4. Bailey, C. P., Bolton, H. E., and Redondo-Ramirez, H. P.: Surgery of the mitral valve. Surg. Clinics of N. Am. 1807, Dec. 1952.

5. Beck, C. S., Hahn, R. S., Leighninger, D. S., and McAllister, F. F.: Operation for coronary artery disease. J.A.M.A., 147:1727, 1951.

directly from the aorta to the coronary vein. There is then a rapid flow of blood from the aorta through the graft into the coronary vein and out into the right auricle while the site of the anastomosis is healing. At a second operation the outflow tract of the coronary vein is obstructed with the result that the pressure of the arterial blood in the vein rises and there is retrograde flow of oxygenated blood through the veins to the sinusoids of the myocardium. This permits increased oxygenation of the heart muscle and has given very promising clinical results.

A congenital condition, the Tetralogy of Fallot, which is one of the causes of the so-called "blue babies" consists of pulmonary stenosis associated with a defect referred to as dextro-position of the aorta. This means that the aorta opens not into the left ventricle, but its opening into the heart is displaced to the right so that the aorta opens both into the left and right ventricles. This prevents the intraventricular septum from being complete at the top. In addition to the obstruction at the outflow of the right ventricle caused by the pulmonary stenosis there is a source of egress of the blood from the right ventricle into the aorta. This condition may be present in varying degrees and it is associated with varying degrees of cyanosis, due of course to the unoxygenated blood from the right ventricle passing into the aorta and thence into the periphery. These patients may die soon after birth or live on into middle life dependent upon the severity of the condition. The surgical correction of this situation may be direct or indirect. Direct in the sense that the pulmonary stenosis or obstruction to the outflow tract is opened much in the same way as described for pulmonary stenosis. This allows more flow of blood to the lungs, reduces the pressure in the right ventricle and thus reduces or stops the flow of blood from the right heart to the left. Another means of improving this situation is based on the fact that much of the unoxygenated blood from the right heart passes into the aorta and not enough blood passes through the pulmonary artery to the lung. Hence if some of the blood from the aorta could be passed back into the lung, the flow of blood through the lung would be increased and the amount of cyanosis appreciably decreased. The operation of Drs. Blalock and Taussig attaches an artery, usually the subclavian, to the pulmonary artery to accomplish this purpose.[6] Another means of doing this is the creation of an opening or "artificial ductus arteriosis" between the aorta and the pulmonary artery conceived by Dr. Willis Potts of Chicago. This is familiarly known as the Potts' Operation for Tetralogy.[7]

Special Preoperative Consideration of Cardiac Surgery

The cardiac patient is prepared for surgery by minimizing decompensation, to give the patient the greatest possible cardiac reserve. This is done by the usual methods

6. Blalock, A. and Taussig, H. B.: The surgical treatment of malformations of the heart in which there is pulmonary stenosis and pulmonary atresia. J.A.M.A. 128:109-202, 1945.
7. Potts, W. J., Smith, S. and Gibson, S. J.: Anastomosis of aorta to pulmonary artery; certain types in congenital heart disease. J.A.M.A. 132:627-631, 1946.

of correcting cardiac failure, such as bed rest, digitalis, diuretics, low salt diet, etc. The patient is evaluated by repeated observations of heart rate, respiratory rate, blood pressure, vital capacity, total body weight and pulse deficit, if present. Important too is physical examination, x-ray, electrocardiogram, venous pressure and circulation time. Aside from the circulation itself, it is wise to test the response of the patient to the planned medications. For this purpose preoperative and operative drugs are given and the patient's response is noted for the ensuing several hours with respect to his pulse, blood pressure, respirations and any other reaction. The drugs we most commonly employ preoperatively are barbiturates, atropine, Demerol (R), quinidine, and Pronestyl (R). We also routinely evaluate the adrenal cortical capacity by the eosinophyl depression test and on this basis decide whether adrenal cortical support will be necessary during and after the operation.[8]

The Choice and Management of Anesthesia

The particular anaesthetics which are used depend upon the personal experience and choice of the anaesthestist. The essential requirements of the anaesthesia are that during the induction and the operation, high levels of oxygenation should be maintained and respiratory acidosis should be avoided. Good control of the anaesthesia level is essential. Anaesthetics that tend to produce myocardial irritability or standstill should be avoided. The events immediately preceding the induction and the induction itself should not excite the patient to start him into tachycardia. Tracheal intubation is of course essential since positive pressure anaesthesia will be necessary. Means for assisted or controlled respiration should of course be at hand. For adults we have used Surital (R) Sodium induction in the patient's room followed by ether anaesthesia with very satisfactory results.

Constant monitor electrocardiography is used and preferably started in the operating room before the tracheal intubation since cardiac arrhythmias may develop at this time. After intubation one may feel quite confident that the airway is clear, but occasionally the tube itself becomes obstructed or a bronchus may not be aerated. This possibility should not be overlooked. In one such case the blown-up bag covered the tip of the intracheal tube and this might have resulted in a fatality if the anaesthetist had not immediately discovered this situation. As the patient is positioned it is important that the chest is not compressed and that the abdomen is free to expand. Particularly in cyanotic children, overheating during operation should be avoided. Cooling with ice bags or other apparatus is often desirable. A large cannula should be placed in a vein, preferably of the ankle, for infusion of fluids and blood as necessary. As the operation progresses, the anaesthetist should note not only the individual observations on the chart, but trends in the chart, because these

8. Blodgett, J. B.: Mitral commissurotomy for multiple valvular disease of the heart. The Grace Hospital Bull.: 30:71, July, 1952.

may anticipate later changes. Changes in the character of respirations, skin temperature changes or cyanosis are of course important to observe. As with other open chest procedures, the lung should be expanded at intervals of about half an hour and the tracheo-bronchial tree be aspirated as necessary. The steps of the operative procedure are followed and noted with the time on the operative chart so that their relation to any change in the patient's condition may be observed and information can be given the surgeon as to the length of time certain steps have required, such as how long the aorta has been clamped. As the chest is closed the lung is carefully and completely reexpanded.

Two emergencies might be mentioned. Under most conditions there is not massive blood loss, but of course in this type of surgery it may and does occasionally occur. The surgeon is grateful for an anaesthetist who immediately starts massive replacement of blood as he is stopping the cause of the flow. Another emergency is cardiac arrest. If this occurs it should immediately be recognized by the anaesthetist on the basis of failure of the pulse, blood pressure, blood pressure oscillations, and cessation of respiration. As you all know the time is short between when the heart stops and cardiac massage to continue the circulation of the blood must be started. The particular contribution of the anaesthetist to recovery of the patient from this condition is early recognition and notification of the surgeon, immediate controlled respiration with oxygen, elevation of the foot of the table to increase venous return, and the introduction of intravenous medications as requested.

After the operation is completed the patient should be changed from the position of the operation gently and slowly and not removed too soon from the operating table, since in the operating room better resuscitation measures may be taken if necessary. As the patient is placed in bed, he is placed in the position of best respiratory exchange and if possible not in the same position as he was in during surgery. He should be turned at frequent intervals to optimal positions for respiration. A chart of pulse, respirations, and blood pressure is kept at fifteen or twenty minute intervals to observe changes in these signs and to recognize trends. Apex and radial pulse rates should be taken at intervals in patients who have auricular fibrillation or in patients who may suddenly fibrillate. If a chest drain has been placed the closed suction drainage should be established promptly to remove any air or blood which has accumulated and also to indicate the amount of bleeding into the pleural cavity. As with all postanaesthetic patients, these people are urged to cough and to take deep breaths at intervals to avoid bronchial obstruction and atelectasis. As to the matter of analgesics, we feel that enough should be given to permit full respiration, cough and moving without excessive pain, but not so much should be given as to considerably depress respiration or the patient's response to requests. Oxygen by nasal tube

may occasionally be indicated. We feel that tents are undesirable because they usually do not deliver as high percentage of oxygen and tend to separate the patient from his attendants. If there is respiratory difficulty on the basis of an organic respiratory difficulty, recognition of this and its correction is much more important than treating the symptom of dyspnoea or cyanosis by placing the patient in an oxygen tent. As to medications, the preoperative medical regime is resumed to support the heart through the early postoperative days until evaluation of functional capacity can be made. Ambulation is permitted as soon as possible but activity should be restricted in cases where rehabilitation must be gradual because of the patient's poor condition preoperatively.

In conclusion the surgical treatment of cardiac patients is a joint undertaking between the cardiologist, the anaesthetist and the surgeon. For the best team function, each must understand the other's problems and objectives. Anaesthesia for these patients applies our knowledge of pharmacology, and the physiology of respiration and circulation. It has given a great assistance toward the progress in rehabilitation of these patients. Certain medical, surgical and anaesthesia techniques and problems have been discussed.

Current Concepts of Pulmonary Function

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Advances in the surgical treatment of diseases of the pulmonary and cardio-vascular systems has necessitated a more detailed understanding of the function of these systems. Surgical treatment is being currently advocated even in patients whose respiratory and cardio-circulatory functional reserve is narrow. It becomes then a matter of fundamental importance to understand, as well as possible, the factors which may determine under what combination of conditions the respiratory and cardio-circulatory reserve is sufficient, or insufficient, to permit safe surgical treatment. It is obvious that this broad subject is also of vital concern to the individual who is responsible for the anesthesia and the maintenance of such a patient during the crucial period of operation.

It is the purpose of this paper to consider pulmonary function in the light of current knowledge and to emphasize such points as seem to have particular application to the problems of the anesthetist.

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It is convenient to consider lung function under two headings; **Ventilation** and **Alveolo-respiration.** The **ventilatory** function of the lungs has to do with the mechanics of moving air into and out of the lungs. The **alveolo-respiratory** phase has to do with the mixing and diffusion of gases in the lungs and the passage of gases from the alveoli to the blood stream and back again.

Ventilation: The average individual at rest moves seven to eight liters of air in and out of his lungs per minute to supply the resting oxygen requirement of about 250 cubic centimeters per minute. Under stress such as exercise, these requirements may increase ten to fifty fold. An elevation in body temperature likewise increases the oxygen requirement significantly. It is important to point out at this time that only about two-thirds of the inspired air actually reaches the basic functional unit of the lung. This is the result of the upper and mid respiratory passages containing 150 to 180 cubic centimeters of dead space before the point is reached at which respiratory gas exchange takes place. The concept of **effective alveolar ventilation** is one of great importance. This amounts to between four and five

liters per minute in the resting state. To accomplish this movement of air, the thorax functions much as a bellows. The descent of the diaphragm and the elevation of the ribs during inspiration results in increased intra-thoracic volume to a degree that the pressure therein becomes a minus 5 to minus ten centimeters of water at the end of inspiration in the resting individual. Air then enters the lungs through the open glottis because of the pressure difference in the atmosphere and that within the thorax in this phase. At the end of inspiration, the diaphragm and thoracic cage return to the resting position and the elastic recoil of the lungs results in sufficient positive pressure to expel the volume of gases inspired. Inspiration then is the active phase of ventilation and expiration largely passive at rest. The amount of air moved in and out during quiet breathing is termed **tidal air** and amounts to about 500 cubic centimeters on the average. It should be mentioned that under the extremes of ventilatory effort, the musculature of the thorax, diaphragm and abdomen are capable of intrathoracic pressures ranging from —80 centimeters of water to plus 100 centimeters of water.

The negativity produced during inspiration is important not only to the inflation of the lungs, but also to the return of blood through the large veins of the thorax to the right heart. The slight positivity of expiration in turn, results in the compression and movement on of the blood already in the great vessels.

One should not neglect to mention the larynx in connection with the ventilatory function of the lungs. This organ is truly the valve of the respiratory system. By fluctuation of the vocal cords, the glottis is narrowed or widened and the changes in intrathoracic pressure so important to ventilation and circulation are influenced. It is perhaps not out of place to mention at this point, the function of the larynx in that important pulmonary defense mechanism, the effective cough. For a cough to be productive, air must be entrapped in the lungs and positive pressure accumulated by the action of thoracic and abdominal muscles against the closed larynx. The larynx then suddenly opens and the accumulated air under positive pressure furnishes the tussive force essential to the raising of pulmonary secretions. Beecher[1] has pointed out the ineffectiveness of cough when an endotracheal tube maintains the patency of the larynx.

A number of technics have been developed to determine the efficiency of ventilation. The term **Vital Capacity** is widely used and may be defined as the amount of air which can be exhaled by maximum effort following a maximal inspiration. The predicted normal vital capacity varies according to sex, age and height, but an average normal figure of about 4,000 cubic centimeters is usually quoted. The measurement of vital capacity is time-honored and still widely used, but it has the shortcoming of measuring volume of ventilation without relation to time; it is

1. Beecher, H. K.: Principles, problems, practices of anesthesia for thoracic surgery. Arch. Surg. 62:203-238 (Feb.) 1951.

static and not dynamic.[2] Certain thoracic abnormalities produce a **restrictive** type of ventilatory impairment. Among these are pleural effusion, pneumothorax, thoracoplasty and pulmonary resection. In these conditions the major impairment of ventilatory function is the restriction of the amount of air which can be moved into and out of the lungs without relation to the time required for this movement. Hence, determination of the degree of diminution of the vital capacity gives a relatively accurate index of the degree of impairment of ventilation under these circumstances. On the other hand, there is an important group of pulmonary abnormalities producing severe ventilatory insufficiency in which the Vital Capacity may be normal. These conditions include bronchial tumors, stenosis, asthma and pulmonary emphysema and produce an **obstructive** type of ventilatory impairment. Given sufficient time to do so, individuals with this type of impairment can move sufficient air past the obstructing lesion to approximate their predicted normal Vital Capacity. It is in this group that the poor correlation between Vital Capacity and pulmonary disability appears. Actually a true evaluation of ventilatory insufficiency depends on a volume-time relationship. The value of the vital capacity determination can be considerably enhanced if the time factor for its performance is considered. It has been found that normally 75% of the vital capacity is exhaled in one second and 95% to 100% within three seconds. The timed vital capacity determination is very sensitive to even the slight ventilatory impairments produced by minimal obstructive defects.

The cornerstone of all tests of ventilatory function is the determination of the **maximum breathing capacity.** This, in contrast, is a test of dynamic function and is determined by measuring the maximum amount of air an individual can move in and out of his lungs in a definite period of time. Its drawback is that complete patient cooperation is necessary. The patient must be able and willing to perform and repeat this rather tiring procedure. Ordinarily this maximal effort is required for only twelve to fifteen seconds. The results are then expressed in liters per minute. Again the predicted normal varies with age, sex and body size. In round numbers, the healthy young woman has a capacity of 100 liters per minute and the healthy young man, 150 liters per minute. This test is very sensitive to air flow resistance as occurring in obstructive defects and also in those conditions of increased turbulence in air flow produced by bronchial secretions.[3] The **maximum breathing capacity** is also highly sensitive to the ventilatory impairment resulting from purely **restrictive defects.**

In mixed forms of ventilatory impairment, information concerning the predominant defect may be obtained by determining the ratio of the percentage of pre-

2. Gray, J. S.; Barnum, D. R., Matheson, H. W. and Spies, S. N.: Ventilatory function tests. J. Clin. Investigation **29**:677, 1950.

3. Proctor, D. E.: Hardy, J. B. and McClean, R.: Studies of respiratory air flow. Bull. Johns Hopkins Hosp. **87**:225, 1950.

dicted normal vital capacity to the percentage of predicted normal maximum breathing capacity performed by the patient.

$$\frac{\%\ \text{Predicted MBC}}{\%\ \text{Predicted VC}}$$

This ratio is designated the **Air Velocity Index.**[4] This ratio is less than one (1) in predominantly **obstructive** defects and one or more in predominantly **restrictive** defects.

An important purpose of pulmonary function testing is to determine reserve which may be required for surgical treatment. Minute ventilation at rest may be determined and subtracted from the maximum breathing capacity giving the breathing reserve. Normally the breathing reserve at rest is equal to 95% of the maximum breathing capacity. Likewise, the breathing reserve may be determined for the individual participating in any activity desired. Application of this principle has been made to a number of practical tests to determine the patient's ability to withstand a planned operation and to predict his functional status post-operatively.

Van Allen and Lindskog[5] and later Churchhill[6] have elucidated an interesting concept of yet another ventilatory phase of lung function. It is proposed that in certain forms of bronchial disease such as bronchiectasis, there is obliteration of some of the finer air passages. It has been shown that though the alveoli of the involved segments have been deprived of their normal ventilatory connections, they remain well filled with air that enters from adjacent normal segments. This **collateral ventilation** is not useful from the point of view of gas exchange in the involved alveoli because it is too slow and because the gas involved has already been through a group of normal alveoli. It does serve, however, the very useful function of preserving the spatial integrity of the lung even after the true ventilatory function of segmental structural units has been destroyed. This is an important concept since the collapse of the diseased segments would necessitate compensatory over distension or emphysema of normal segments to meet the spatial demands of an air-containing organ enclosed in a cavity with more or less rigid walls. This would be of considerable functional disadvantage to the normal segments as will be discussed later.

The **alveolo-respiratory phase** of lung function is concerned, as was mentioned earlier, with the mixing and diffusion of gases in the lungs and the passage of gases from the alveoli to the blood stream and back again. A factor in the mixing and diffusion of gases in the lung is the relative size of the residual air compartment compared to the total lung capacity. The vital capacity may be considered here to represent that portion of total lung capacity which can be measured directly. At the end of the maximal expiration used to determine vital capacity, there remains in the lungs of everyone a significant volume

4. Gaensler, E. A.: Air velocity index. Numerical expression of the functionally effective portion of ventilation. Am. Rev. Tuberc. **62**: 17-28 (July) 1950.

5. Van Allen, C. M.; Lindskog, G. E. and Richter, H. G.: Collateral respiration. J. Clin. Investigation **10**:559, 1931.

6. Churchill, E. D.: The architectural basis of pulmonary ventilation. Ann. Surg. **137**:1, 1953.

of gas known as the **residual air** or **residual capacity.** Normally, this amounts to 20% to 25% of the sum of the vital capacity and the residual capacity which, in turn, equal the total lung capacity. Since the gases of the residual air compartment do not move freely in and out of the lung, it follows that they act to contaminate the air that is brought in to a greater or lesser degree, depending on the size of this compartment. In other words, the air remaining in the residual compartment is high in carbon dioxide and low in oxygen and the fresh air brought in must mix with the residual air in the process of reaching the alveoli. In certain diseases such as pulmonary emphysema and bronchial asthma, air is trapped in the alveoli resulting in over distension of the alveoli and a definite increase in the residual capacity. The more advanced this condition becomes, the poorer the intrapulmonary mixing and the greater the contamination of inspired air. The patient becomes more and more disabled from an alveolar-respiratory insufficiency. The residual capacity may be measured indirectly by washing out and collecting the nitrogen from the residual compartment. This is accomplished by having the patient breathe oxygen for seven minutes. At the end of the procedure an alveolar gas sample is secured by collecting the very end of a forced expiration. Analysis of this sample for nitrogen furnishes another important index of intra-pulmonary gas mixing. Normally the concentration of nitrogen in this sample is less than 2.5%.

Patients in the older age group are prone to have some degree of emphysema and hence impaired **alveolo-respiratory** function. There is a tendency here toward ineffective alveolar ventilation with arterial anoxia and hypercapnea. Because of chronic exposure to high carbon dioxide, the respiratory center becomes relatively insensitive to this gas. The stimulus to ventilation is operating through the carotid receptor mechanism. These patients present a distinct problem to the anesthetist if surgical treatment is necessary. Often a degree of cyanosis will indicate that high oxygen gas mixtures are indicated. Yet as the anoxia is improved, the stimulus to ventilate, mediated through the carotid body by oxygen lack, is removed. The patient may ventilate very poorly with further accumulation of carbon dioxide and increasing respiratory acidosis or may actually stop breathing. It is essential that effective alveolar ventilation be maintained in these patients. The oxygen is necessary, but in addition adequate ventilation must be maintained by artificial methods such as manual compression of the anesthesia bag. Provisions must be made for continuing artificial respiration in the post-operative period because the severely emphysematous patient simply will not spontaneously breathe well in a high oxygen environment.

The final aspect of lung function to be considered here is the matter of respiratory gas exchange between the alveolus and the blood stream. The partial pressure of the gases on each side of the alveolo-capillary membrane

is the major factor determining the diffusion of gases between the blood and the alveoli. In general it may be said that carbon dioxide is more soluble than oxygen and hence crosses the alveolo-capillary membrane more easily than oxygen. The carbon dioxide tension is the same in the peripheral arterial blood as in the alveolus. It is fundamental to recall at this point that there is a reciprocal relationship between effective alveolar ventilation and alveolar carbon dioxide and hence blood carbon dioxide. If effective alveolar ventilation is not maintained in the anesthetized patient, alveolar and blood carbon dioxide will rise and respiratory acidosis will ensue. An index of the efficiency of gas exchange between the alveoli and the blood stream is obtained from analyzing peripheral arterial blood. At rest, the arterial oxygen saturation is 95% or above and the pCO_2 40 millimeters of mercury. Normally these values do not change after exercise and indicate an adequate gaseous exchange. If the carbon dioxide partial pressure increases, the cause of the difficulty is poor alveolar ventilation. This situation is encountered in poliomyelitis, emphysema and often in patients during operations requiring the opening of the chest. Arterial oxygen desaturation may be the result of the admixture of venous blood which occurs when a group of alveoli are perfused with blood but not ventilated. Pulmonary atelectasis produces this sort of situation. Thickening of the alveolo-capillary membrane as in pulmonary edema also markedly interferes with the diffusion of oxygen between the alveolus and the blood stream.

It would seem justified to close this presentation by emphasizing certain aspects of pulmonary function that apply significantly to the problems of the anesthetist. In the first place, the problem of insuring adequate removal of carbon dioxide is a pressing one. Serious acidosis due to inadequate alveolar-ventilation may occur and is particularly prone to be present in thoracic operations. This acidosis can be completely prevented by assistance to ventilation. This ventilation must exceed values considered adequate for the normal conscious individual. In this connection it should be recalled that the respiratory passages contain 150 to 180 cubic centimeters of dead space and that this figure should always represent less than one third of the total ventilation if adequate alveolar ventilation and carbon dioxide elimination are to be attained. It should be remembered that any positive pressure makes elimination of carbon dioxide more difficult. Positive pressure must be used in assisting ventilation, but should be completely relaxed during the expiratory phase to facilitate removal of carbon dioxide. The induction period, as well as the periods of intubation and suction are those during which hypoxia is prone to develop. Particular care should be exercised in the suction of the endo-tracheal tube. Prolonged suction produces profound hypoxia and hence, suction should be applied only for short periods interspersed with periods of high oxygen administration.

It should be emphasized again that the patient with pulmonary

emphysema presents a difficult problem in anesthesia in any type of surgery. These patients are very prone to develop severe respiratory acidosis. Oxygen is necessary, but administration of high oxygen mixtures eliminates the carotid body activity upon which these patients depend for the stimulus to breathe. Hence, very careful and constant attention to artificial ventilation must be maintained. It may not be out of place to mention here that in this type of patient with increased pCO_2 and acidosis, the conduction of cardiac impulses is impaired. There is a tendency toward ventricular tachycardia and an increased sensitivity of the heart to atropine. Carbon dioxide retention is probably as important in the etiology of cardiac arrest as is hypoxia. It is important to remember that there is no absolute method to recognize carbon dioxide retention, but it may always be prevented by adequately ventilating the patient.

It is my opinion that continuous positive pressure anesthesia has no advantage to offer. It impairs elimination of carbon dioxide and seriously interferes with the return of blood to the right heart. Periodic inflation of the lung during thoracic procedures and assistance to ventilation may be secured by positive pressure during the inspiratory phase alone.

Summary

The functions of the lungs have been reviewed and methods of testing these functions presented. An effort has been made to emphasize those facets of this subject which have special application to anesthesiology.

Factors In Achieving Satisfactory Endotracheal Anesthesia

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Endotracheal anesthesia is assuming more importance every day, until it is necessary that anyone who assumes the responsibility of the health and life of the anesthetized patient understand and be capable of utilizing this method, both in a planned procedure and in an emergency.

One may regard an endotracheal tube as merely an extension or prolongation of the trachea, so that an air-tight system is effected between the lungs and the anesthetic machine. This makes possible easy regulation of the gaseous content of oxygen, carbon dioxide and anesthetic vapors in the lung. It likewise provides a water-tight system to exclude extraneous matter and at the same time facilitate aspiration of pulmonary secretions. It affords complete control of the respiratory motion of the lungs and thereby their ventilatory function regardless of the status of the chest wall—whether flail from a thoracoplasty or trauma, or where widely open by surgical design through the rib cage or the diaphragm.

The usual indications therefore, for endotracheal anesthesia are first of all intrathoracic or transthoracic procedures where it is the only practical method of maintaining adequate respiration. Where bronchial secretions are a prominent factor as in bronchiectasis or lung abscess, whether the surgery be pulmonary or otherwise, the endotracheal tube affords a ready means of aspirating the secretions. By thus keeping the tracheo-bronchial tree dry, a satisfactory anesthesia can usually be maintained without periods of serious hypoxia and without the complications of atelectasis or pneumonia due to retained secretions.

Intubation becomes a great convenience in head and neck surgery by allowing both the surgeon and the anesthetist to ply their trades unmolested where otherwise they would be competing for the same anatomic terrain.

In upper abdominal surgery controlled respiration may be of great value technically to the surgeon. During a light anesthetic, mesenteric traction or diaphragmatic stimulus will incite cough and laryngeal spasm which may be prevented by having the endotracheal tube maintain an open larynx. This method may be much preferable to the very deep anesthesia that would otherwise be required.

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When gastric secretions or ingested food may be dangerous, as is so often true in emergency work, the endotracheal tube adds a safety factor by excluding foreign matter from the trachea while providing a ready means of aspiration. In addition it is wise to induce vomiting prior to starting the anesthesia.

Anatomic deformities of the head and the neck that prevent a tight fit of the mask or cause obstruction by the tongue or larynx may be circumvented most readily by the endotracheal tube.

All anesthetists are fully aware of the importance of maintaining a proper depth of anesthesia and know how this may be achieved. However, all too often the importance of maintaining complete oxygenation and of adequately removing carbon dioxide is less well appreciated. Periods of apnea or shallow breathing cannot be observed tolerantly with the expectancy of an early return of adequate spontaneous breathing by the patient. Oxygen is the most vital of all substances required to maintain life and its deprivation for more than a very few minutes will severly damage vital structures, often sufficiently to prove fatal. Shorter periods of deprivation lead to respiratory depression, pulmonary edema and general deterioration of the patient's condition. Similarly, interference with elimination of carbon dioxide may prove fatal or markedly disturb the physiology of the body as respiratory acidosis is produced. Cerebral damage is directly proportional to the degree of interference with gaseous exchange. The brain centers assume lower levels of activity while certain organs such as the heart become much more susceptible to abnormal function. Cardiac arrest, while occasionally due to drug toxicity, is most often due to the increased myocardial irritability that follows hypoxia and hypercapnea. Thus, whenever respiration is impaired by drugs, anesthetics, position of the patient, the open chest or high abdominal surgery, it is absolutely incumbent on the anesthetist to achieve and maintain at all times adequate respiratory exchange. This must be judged by the tidal volume of the patient and not merely by the respiratory effort.

The technic of intubation is not difficult and is readily learned with a little practice. There are again certain principles which aid in making it an easy procedure. First of all adequate relaxation of the jaw muscles is essential. The safest method of achieving this is by a relatively deep anesthesia. Ether has definite advantages in that it stimulates respiration and by allowing only slow alterations in the depth of anesthesia, it affords a longer safe working period for the intubation. The paralyzing drugs, the curare derivatives, are of value when necessary but are in fact rarely needed and they add to the hazards of the procedure by further reducing the patient's own respiratory efforts and thus encouraging asphyxia. Prior to the intubation one should ascertain that the patient is well oxygenated—assisting respirations wherever necessary. If one attempt is made at passage and is unsuccessful, the mask should immediately be replaced on the face,

the patient reoxygenated and the proper depth of anesthesia reattained before any second attempt is made. This will help obviate long periods of anoxia, a hurried traumatic instrumentation, or laryngeal spasm secondary to manipulations at light levels of anesthesia. Laryngeal spasm may be produced by placing the laryngoscope too close to the larynx even without touching the cords, or by a clumsy, misdirected thrust of the endotracheal tube even in the presence of a deep general anesthesia or a complete topical anesthesia. Occasionally it is helpful to insert the endotracheal tube under topical anesthesia in the conscious patient and then administer the anesthetic with complete control of the airway from the very start.

The technique itself involves primarily maintaining the neck straight or slightly flexed with hyperextension of the head on the neck at the atlanto-occipital joint. The laryngoscope is inserted to one side of the tongue so that the entire base of the tongue will not have to be lifted. The jaw is lifted forward and the epiglottis elevated out of the field. If the scope is then lifted—not rotated, which would apply pressure to the front teeth—the vocal cords can be visualized. The endotracheal tube is then dextrously inserted during inspiration under direct vision. We should not forget that endotracheal tubes may be passed blindly through the nose or through the mouth, should such be desired. Most of all, remember that introduction through a tracheotomy is extremely easy and very helpful where there be extreme jaw or neck deformities, or mutilating injuries of the head and neck in emergency cases. If the nature of a deformity or the operation itself is going to necessitate a tracheotomy for the post-operative care, it will usually be desirable to perform the tracheotomy as the first part of the surgical procedure and administer the anesthesia through this.

The tracheal tube must of course be inserted past the vocal cords but not so far as to pass into one or the other main stem bronchus. The trachea is approximately four or five inches in length in the average adult. A safe guide is to pass the endotracheal tube about one-half inch past the vocal cords which can be gauged at the time of intubation by observation through the laryngoscope. Other safeguards include having the total length of the endotracheal tube so short that it will not reach to the carina even if inserted to its full length. After passage of the tube, inflation of the chest by compression of the anesthetic bag should reveal full equal motion of both sides of the chest. In addition, ausculation of the chest with a stethoscope will reveal the presence of breath sounds on both sides, indicating complete patentcy of the bronchi to both lungs. It is important to check the position of the tube again after positioning the patient, if any postural change is made. The endotracheal tube cuff is inflated until a moderate pressure on the anesthetic bag will produce no leakage of air around the tube in the trachea. Excessive inflation of the cuff may cause necrosis of the tracheal mucosa while too

little will not produce an air-tight system. Should the cuff break, a new tube may be passed. However, if for any reason this is not desirable, a fairly air-tight system may usually be obtained by packing the pharynx with moistened gauze. Similarly it is quite feasible to leave the tube in the trachea and place a tight mask over both the face and the open end of the endotracheal tube. This will still permit positive pressure anesthesia and also afford access for suctioning as desired.

Bronchial secretions should be aspirated before positioning the patient and before the surgery is started. One is nearly always surprised at how frequently something may be aspirated in a relatively dry patient. The practice of listening to the connecting tubes for sounds indicating moisture is extremely misleading as untold amounts of foreign matter may be present in the bronchi without producing auscultatory evidence for the observer listening to the tubes, or for that matter even listening to the chest itself with a stethoscope. During the introduction of the aspirating catheter through the endotracheal tube, no suction should be applied as this unnecessarily prolongs the period of aspiration, removing however, both oxygen and anesthetic vapors from the lung. The period of applying suction should be brief. The patient is then reoxygenated and later reaspirated, repeating this cycle as often as is necessary. We have frequently observed a prolonged period of aspiration drop the oxygen saturation of the blood from a normal of 95% to 70% or less. I need not again emphasize the dangers of this hypoxia which may lead to cardiac arrest, cerebral damage, pulmonary edema or other distressing complications.

In any case in which the chest is open the respiratory effort by the patient draws air through the wound as well as through the trachea into the lungs. The relative amounts of air passing through the trachea and through the chest wound are roughly proportional to their respective diameters. As the chest incision is infinitely larger than the trachea, it can be readily appreciated that it is usually impossible for the unassisted patient to maintain adequate respiratory exchange. It is true that the mediastinum in the human is relatively stable but usually not sufficiently so as to prevent the atmospheric pressure from being transmitted to both lungs. If the patient be lying on his side, the lower lung not only has the transmitted atmospheric pressure but also the weight of the heart and other mediastinal structures to support, and even lift during inspiration. Many methods have been used in order to combat the atmospheric pressure. Among the first was constant insufflation of oxygen down the endotracheal catheter. This was fairly effective in maintaining oxygenation but was not at all satisfactory for removing carbon dioxide. Subsequently a closed system with constant positive pressure of 8 to 10 cm of water was used to neutralize the atmospheric pressure. This usually allowed satisfactory inspiration but handicapped markedly the expiratory effort by the patient, so that adequate oxygenation was

often accomplished but the removal of carbon dioxide was impaired with resultant respiratory acidosis.[1] In addition, the increased intra-alveolar pressure tends to compress the pulmonary capillaries, preventing the blood flow through the lungs and thus venous return to the heart. This of course decreases the cardiac output and if prolonged is often followed by a fall in blood pressure.[1] [2] This is most marked of course in the patient who is already in poor condition.

In recent years we have come to realize that a much more satisfactory method is that of an intermittent positive pressure, utilizing the increased pressure only during inspiration and allowing the patient to exhale against atmospheric pressure. It has likewise been demonstrated by Maloney and others[3] that even the intermittent positive pressure has a mild deleterious effect on pulmonary blood flow and cardiac output, due to the same, though less pronounced, interference with venous return to the heart. Because of this, intermittent positive pressure respiration is much inferior to a system of alternating positive and negative pressure respiration, which actually aids venous return and maintains circulatory dynamics. At the present time however, there is no generally available adequate method of providing the intermittent positive-negative pressure type of respiration, so that we rely on intermittent positive pressure which in general is very efficient. Intermittent positive pressure is usually furnished by repeated manual compression of the rebreathing bag, though some have devised mechanical respirators for this purpose. A tidal volume of five or six hundred cc approximately 20 times a minute will assure any normal patient adequate oxygenation and removal of carbon dioxide. This is true regardless of the position of the patient.

Usually assisted respiration is all that is necessary. During this the bag is compressed with each inspiration to augument the inspiratory volume, but allowing the patient to maintain his own respiratory rhythm. Often however, it is preferable to take over breathing completely for the patient and establish a rhythm and periodicity of breathing completely subject to the volition of the anesthetist. This controlled respiration can be accomplished by paralyzing the respiratory muscles by curare derivatives. A safer method, however, is to deepen the anesthesia and assist the respiration sufficiently to lower the carbon dioxide level and assure oxygenation of the patient. This removes the usual respiratory stimuli and during the ensuing apnea the anesthetist may establish the rhythm and depth of respiration desired. Normal respirations will usually resume as the anesthesia is lightened.

At the end of the procedure as the chest is being closed, careful attention must be paid to complete reinflation of the lung and to vigorous aspiration of the tracheo-bronchial tree. During this period of course, as always, it is important to maintain the oxygenation of the patient. It is easy to become over enthusiastic in removing the secretions and to forget the respiratory needs of

the patient, leading as a consequence to the cardiac arrests which have been reported to occur during the period of extubation.

Summary

Endotracheal anesthesia is merely the adaptation of a tracheal prolongation to usual sound anesthetic practices. The technic of placing the endotracheal tube is relatively simple and when used with the above mentioned precautions is quite safe. In addition to the usual anesthetic management, should respiration be compromised in any manner the maintenance of an adequate respiratory exchange—breath by breath—becomes the responsibility of the anesthetist.

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Post-Operative Recovery Room

The Anesthetist's Responsibility

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The immediate post-operative and post-anesthetic period is without a doubt the most dangerous one for the surgical patient. It is at this time that most of the fatalities, incident to the operation, occur. There are a number of reasons for this. One of the foremost is that the vigilance of the surgical team is relaxed at this time. Another is that at this time, the patient is frequently entrusted to less skilled individuals. The patient is returned to his bed and, either because of the scarcity of personnel or other reasons, is left alone at this most crucial time. During the period of awakening, the possibility of vomiting with its catastrophic aspiration is ever present. Circulatory changes which do not manifest themselves during anesthesia and surgery are frequently precipitated at the conclusion of anesthesia. Shock in its various forms may be first manifested in the post-operative period. The changes in blood pressure and pulse attending the discontinuance of anesthesia or removal of a patient from one position to another or from the table to the roller and to his bed, is known to all who care for surgical patients. Surgeons, anesthesiologists and nursing supervisors all recognize the need for more and adequate supervision of surgical patients, immediately after operation. Shortage of nursing personnel has focused attention towards this problem and brought it to a climax. As a result the post-operative recovery room has evolved. This not only permits the concentration of all the acutely ill patients who have undergone operations into one locality but it also permits to a fuller extent the utilization of more expert help in the care of such patients. In most institutions, the post-operative recovery room is a cooperative endeavor, participated in by the surgeon, the anesthetist and the surgical nurse.

The majority of complications and fatalities in the immediate post-operative period are either due to asphyxia or to irreversible shock. Among the less common causes for fatalities are emboli, coronary occlusion, respiratory failure from a central lesion, such as are seen in head injuries or brain tumors, cerebral hemorrhage, uncontrollable hemorrhage and transfusion reactions. Many of these deaths are preventable, particularly those due to asphyxia.

Complications due to asphyxia

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are largely the concern of the anesthetist. The anesthetist's responsibility ends only when the patient is no longer in danger from asphyxia, anoxia or carbon dioxide excess. The responsibility of the anesthetist extends, then, from the moment the patient is anesthetized until the patient has completely recovered his reflexes and is fully awake. The period of responsibility does not end as has heretofore been the custom, at the moment the patient is removed from the operating room, but rather throughout the period in which the patient is unconscious and is unable to care for himself in the event vomiting or obstruction occurs. Patients who develop respiratory failure from intracranial disease and require artificial respiration for a protracted period of time are also the concern of the anesthetist as well of interest to the surgeon. Irreversible shock, of course, is more the concern of the surgeon than the anesthetist and in many cases is an excusable cause of death.

The responsibility for patients in the recovery room is shared by the anesthetist, the surgeon and the surgical nurse. The surgeon attends to the surgical aspects of the patient's care. The surgical nurse provides the general nursing care, she administers the antibiotics, sedatives, catheterizes the patients and performs other such duties as requested by the surgeon. The anesthetist principally cares for the airway and removal of secretions from the tracheal bronchial tree. It is her duty to see that anoxia or carbon dioxide excess are avoided and to assist as much as possible in watching the circulation and the management of fluid therapy.

In a large hospital such as Charity Hospital, where approximately 90 surgical patients are operated upon daily, a large recovery ward is necessary. The recovery ward should in all instances be placed as close to the operating room as possible in order to be accessible to both surgeons and anesthetists. In hospitals where anesthesiologists are available to head the department of anesthesia, the physician anesthetist outlines the routines and technics to be followed by members of the anesthesia department in the recovery unit. At the Charity Hospital where a school for the training of nurse anesthetists exists, it is feasible to assign a nurse anesthetist to remain constantly in the recovery room during the peak hours of surgery. These peak hours are from 9 a.m. to 5 p.m. on week days. During the evening the nurse anesthetists on call visit the recovery room periodically. The recovery room is in charge of one of the anesthesia residents who makes rounds periodically, usually at hourly intervals throughout the twenty four hour period. Members of the anesthesia department and the surgeons confer with each other to decide which patient is to go to the recovery room. In general all patients undergoing major surgery, regardless of the type of anesthesia should be sent to the recovery room for a period of observation. In certain uncomplicated cases, exceptions may be made for those who were operated under local anesthesia and for those orthopedic patients who are to be placed in traction immediately. Pediatric patients, despite

the objections of pediatricians, are best cared for in the recovery room since equipment for resuscitation is available and since the recovery room is accessible immediately to both the surgeon and the anesthetist.

If the surgeon feels it is not necessary for the patient to remain in the recovery room, it is customary to indicate on the post-operative orders the following: "Patients may be removed from recovery room as soon as released by a member of the Anesthesia Service." This means that the Anesthesia Department has not discharged its responsibility and feels that the patient should be continued to be watched for obstruction, vomiting and other complications which might ensue before reflexes have returned. As soon as the patient is free from these dangers, he may be transferred to his ward or room. In cases where the operation has been complex or formidable, such as in cardiac surgery, pulmonary surgery, gastric surgery or neuro surgery, where the surgeon wishes to watch the patient's progress and administer special treatments, such as blood, plasma, aspiration of the chest, tidal drainage, etc., or have special care administered, it is indicated in the post-operative orders. The order is written as follows: "The patient is not to be removed from the recovery ward until released by both anesthetist and surgeon." In these instances it is not uncommon for the Anesthesia Department to release the patient within an hour but the surgeons retain him in the recovery room for longer periods of time. In smaller institutions the recovery room is operated during the day, usually from 8:00 A.M. to 4:00 P.M. In recovery rooms in smaller hospitals which are not open on a 24-hour basis, the combination roller bed facilitates closing the unit at the end of the day. In larger institutions the recovery room operates on a 24-hour schedule.

Equipment and supplies necessary for the post-anesthetic care of the patient is supplied by the Anesthesia Department. These should include airways of various sizes, laryngoscopes of different type; including the McIntosh, the Guedel and a small Miller blade for infants. A handle with interchangeable blades is the best. Various types of endotracheal tubes are also held in readiness as well as a bronchoscopic blade to fit the laryngoscope. Equipment for administration of oxygen including nasal catheters and positive pressure masks are also assigned to the recovery room. In addition oxygen tents for infant use are necessary. In hospitals where it is feasible, tents may be used for adults. A resuscitator of the blow and suck type is available as well as carbon dioxide oxygen mixtures for inhalation. Analeptic drugs used for stimulation likewise should be supplied by the Anesthesia Department. These include metrazol, coramine, picrotoxin and allylnormorphine. The metrazol and coramine are used for depression of various sorts. The picrotoxin is used in event of barbiturate depression. The allylnormorphine (or Nalline) is used for morphine and other narcotic overdosage. In addition stimulating drugs that raise the blood

pressure are available. These consist of drugs such as arterenol and ephedrine. Besides the stimulants, an intravenous barbiturate likewise should be available for the control of convulsions due to local anesthetic reactions or other causes. Seconal or nembutal is the most desirable for this. Dramamine for parenteral injections and phenobarbital to control nausea are desirable. The suction apparatus is one of the most important pieces of equipment used by the anesthetist in the recovery room. Copious secretions are common after operations. These are best controlled by suctioning. Catheters and metal pharyngeal suction tips should be available. As patients are emerging from anesthesia, a certain degree of spasticity of the jaw muscles is present. The catheter is ineffective in removing secretions from the mouth in these circumstances. A stiff metal suction tip is preferable. Drying agents such as atropine or scopolamine are necessary for cases when secretions continue to form. Apparatus for the use of positive pressure in the event of laryngeal spasm is necessary. It is not uncommon for patients to develop pulmonary edema in the post-operative period. Here again positive pressure is indicated. An iron lung should be available but it is not necessary to be kept in the recovery room. It is used when a patient develops apnea, which is prolonged, such as is found after brain operations. A patient placed in the iron lung is cared for by the anesthetist from the standpoint of airway and oxygen therapy.

It is obvious that smaller hospitals cannot afford or obtain anesthesia personnel who remain in the recovery room constantly. In addition the volume of surgery is not sufficient to warrant such a staff. In such instances the chief anesthetist, whether nurse or doctor, assigns a staff anesthetist to remain with the patient until the immediate danger is over. Then the care of the patient is turned over to the surgical nurses. In such instances, it is the responsibility of the Anesthesia Department to instruct and train surgical nurses in the management of the airway and in being able to recognize such complications as may arise pertaining to anesthesia so that they may immediately summon help. The Anesthesia Department then makes rounds periodically at convenient times and decides the condition of the patient and determines the time he may be discharged from the recovery room. Details such as suctioning the patient, the administration of oxygen, etc., are supervised by the Anesthesia Department. Prescription and the recommendations for the uses of stimulants and fluids in the absence of a physician specializing in anesthesia are then the responsibility of the surgeon. The nurse anesthetist assists in carrying out the treatments and does not assume the responsibility for ordering treatments. After inhalation or basal anesthesia, the patient is not released from the recovery room until vomiting has stopped and the blood pressure and pulse have become stabilized, a free airway is present and complete recovery of all his reflexes has occurred. This latter is a very

important feature, because in the event a patient regurgitates or slips back into narcosis his airway may become obstructed and he will die from asphyxia. After spinal anesthesia the patient may be safely removed to his room when the paralysis has disappeared. It is not an uncommon occurrence to have the blood pressure fall, usually from primary shock after the block has lost its effect. In these cases, if the patient is not being watched, serious circulatory derangements may occur. No patient should ever be removed from the recovery ward who has been brought there with an endotracheal tube or artificial airway in place, until he can fully maintain his own airway unassisted by any artificial device. Obviously the less the patient is moved about or shifted from table to carriers and carriers to bed, the better for the patient.

In conclusion then, the anesthetist's responsibility for anesthesia extends beyond the operating room to the bedside and does not end until the patient is no longer in danger from asphyxia or other respiratory difficulties. Treatments and care other than those pertaining to anesthesia are the responsibility of the surgeon and the surgical nurses.

Rectal Pentothal For Cardiac Catheterization In Children

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In the majority of children on whom cardiac catheterization is performed, oral medication with one of the barbiturates and local infiltration of the site of venisection is sufficient to ensure cooperation. More profound narcosis is necessary for the uncooperative child or for those whose antecubital veins are of such small caliber that catheterization must be done by means of the saphenous vein.

Certain criteria are necessary in the agent used for this procedure: (1) It must be non-explosive, (2) it must be under control during periods of darkness while the fluoroscope is being used, and (3) oxygen saturation levels of the blood must not be appreciably altered.

Burnap et al[1] have used rectal pentothal for catheterization of the heart in children. Marbury[2] found that blood oxygen saturation was not greatly changed in children under rectal pentothal. We have used pentothal rectally for the past five years for basal hypnosis in all children with congenital heart disease and other selected pediatric cases with gratifying results. It was decided to extend its use to those catheterization cases in which anesthesia was required.

This report is based on 6 cases of rectal pentothal for cardiac catheterization on uncooperative children or small children whose saphenous veins were catheterized. Preparation: The child is weighed on admission to the ward. A cleansing saline or tap water enema is given early in the evening. Atropine sulfate in dosage suitable to the age and weight is given 30 to 45 minutes before the child is brought to the cardiopulmonary laboratory.

Dosage of Pentothal: Dosage is computed 1. Gm. /50 lbs. or 20 mg. per lb. of body weight as recommended by Weinstein and Light.[3] The solution is freshly prepared for each patient. A 10% solution is prepared by using 10 cc. of tepid tap water to each Gm. of Pentothal. 1 cc. of this solution contains 100 mg. of Pentothal. We have found that the use of a 10% solution facilitates ease of calculation of the dosage since

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1. Burnap, R. W.; Gain, E. A., and Watts, E. H.: Basal anesthesia in children using sodium pentothal by rectum. Anesthesiology 9:524-531 (Sept.) 1948.

2. Marbury, B. E.: A study of the blood-oxygen saturation in children under rectal pentothal anesthesia. Anesthesiology 11:589-591 (Sept.) 1950.

3. Weinstein, M. L., and Light, G. A.: Rectal sodium pentothal in 2,500 cases. Anesth. & Analg. 27:343-350 (Nov.-Dec.) 1948.

the simple multiplication of the weight in pounds by .2 will give the number of cc's of the 10% solution to be used. The use of a concentrated solution also makes it necessary to give a smaller volume thus aiding retention.

Procedure: The child is brought to the cardio-pulmonary laboratory by a nurse who is trusted by him in order to avoid emotional disturbances. The rectal instillation of the pentothal is done in the ante-room of the laboratory rather than the patient's room to assure utilization of the entire period of the drug's effectiveness. A child will willingly submit to the insertion of a #10 or 12 Fr. catheter into the rectum after being reassured that he is just going to have his temperature taken. The calculated dose is injected with a Luer-lok type syringe, allowance being made for the amount of solution necessary to fill the catheter. First signs of drowsiness appear in 10 to 15 minutes. When optimum signs of narcosis are evident the patient is gently transferred to the fluoroscopic table, electrocardiograph leads are applied and the site is prepared for catheterization. Procaine 1% is injected over the vein to be catheterized.

A laryngoscope and intratracheal tubes are kept in readiness at all times, as is a machine for the administration of oxygen. It has not been necessary to resort to the use of these in any of the cases done so far. Signs of obstruction of the airway or of anoxia have not been seen. Although there is a slight decrease in depth of respiration when no stimuli are being applied, no instance of significant respiratory depression was experienced. Application of stimulation caused movement of the head or extremities but were not sufficiently troublesome to interfere with the procedure. There have been no cases of post-anesthesia delirium or vomiting. Most of the children responded at the end of the procedure. Electrocardiographic evidence of irregularities have been present only with the passage of the catheter into the heart and were not attributed to the anesthetic.

In only one case was it necessary to supplement the rectal pentothal. This was in an extremely apprehensive 10 year old girl weighing 120 lbs. on whom catheterization via the saphenous vein was done. This child required small additional amounts of intravenous pentothal solution to control restlessness. In this case supplemental oxygen was given although there were no apparent signs of anoxia.

Summary and Conclusions

The use of rectal pentothal solution for cardiac catheterization in 6 children is reported. Onset of hypnosis was pleasant. Rectal pentothal is safe to use with the fluoroscope both from the standpoint of non-explosibility and darkness. No complications have been observed. Blood oxygen saturation samples have not been altered. The effects of the pentothal wear off by the close of the procedure, thus obviating the need for long post-anesthesia observation. In cases of cardiac catheterization in children where hypnosis is required we feel that rectal pentothal serves as a satisfactory agent.

We wish to thank Dr. Henry A. Zimmerman, M.D., Chief of the Cardio-pulmonary Laboratory, for his counsel and assistance.

Legislation

Anesthetist Liable For Injuries Caused By Use Of Anesthetic Contrary To Patient's Authorization—A case which was tried on the theory of an assault by reason of the use of a method of anesthetizing objected to by the patient resulted in a verdict of $60,000 for the patient against the anesthetist.

The family doctor had diagnosed the patient's condition as a chronic appendix which should be removed. He referred her to a surgeon who confirmed the diagnosis. The patient asked who would administer the anesthetic and the surgeon told her it would be done by a certain medical anesthetist. She stated that she was afraid of a spinal block, whereupon the surgeon informed her a general anesthetic would be administered.

The surgeon instructed the hospital to write on the chart that the patient did not want a spinal anesthetic. After the surgeon entered the operating room he discovered that the patient had been given a spinal anesthetic. Although the anesthetist noted the statement in the chart of the patient's aversion to a spinal anesthetic he saw nothing in her condition which would contraindicate the use of a spinal block. He discussed it with the patient while she was on the operating table and she consented. He introduced the spinal needle into the fourth interspace and injected the anesthetic. He and the nurse then placed the patient on her back, laid her right arm out on the table and injected sodium pentothal into her arm and she was put to sleep. The patient, however, denied that she had consented to the use of the spinal anesthetic.

At the trial a medical doctor, who qualified as an expert witness for the patient, based upon his examination of her and a study of all her hospital records and history, was of the opinion that the patient had sustained a puncturing of the spinal cord which had resulted in an active inflammation of the spinal cord, producing partial paralysis from the waist down, ankylosis of the hip joints, complete loss of power in the feet, shrinking of the legs, and migraine headaches.

In his own behalf the anesthetist testified that the patient had a normal convalescence until the sixth day when she developed definite indications of nerve disturbance due to having contracted polio. Several medical experts testified likewise that the paralytic condition of the patient was caused by polio and that the spinal block was not a contributing factor thereto.

The court held, first, that the surgeon was not liable. His agreement with the patient that she would not be given a spinal block meant that he would do everything reasonably to be expected to see that she was not given one. He put the instruc-

tions on the chart, intending that the anesthetist should read and observe them, and the anesthetist did read them. The surgeon did everything that could be reasonably expected he would do and was obliged to do. Since the anesthetist was an expert in the administration of anesthetics, he could not be considered the surgeon's agent in the method of administering the anesthetic.

As for the anesthetist, as he used a method he was told by the patient not to use he was an assaulter. The evidence of the patient was, by reasonable inference, that he did read the record and did know of the patient's demand that no spinal block be administered. The jury's finding that she had not consented is binding upon the court, as was the jury's conclusion that the injuries were the proximate result of the assault.

(Woodson v. Huey, 2 CCH Neg. Cases 2d, 284-Oklahoma Supreme Court, June 23, 1953)

Death of Newborn By Bassinet Fire Due to Professional Act of Negligence—A student nurse in charge of a nursery, on noticing that a baby's feet were cold and his temperature less than normal, bent a gooseneck lamp over its bassinet so that its lighted 100 watt bulb, with the reflector, was about three inches from the blanket, directly over the baby's feet. The placing of the bulb too near the blanket resulted in a fire and the baby was burned to death.

The hospital was held not liable for the nurse's negligence, because the act was performed as medical and professional care, and not merely as an administrative act. No testimony was offered to show that the procedure followed by the hospital in its care for newborn infants was not the standard of care employed by other hospitals in the city. No defect was found in the lamp, it was an ordinary lamp with an ordinary electric light bulb, and an ordinary reflector, used on prior occasions over a period of four years.

(Cadicimo v. Long Island College Hospital, Sup. Ct. Kings Co., Trial Term Part V, Arkwright, J., N.Y.L.J., May 14, 1953, p. 1627)

Infant Awarded $55,000. Against Hospital For Failure To Provide Proper Care For Mother—The patient alleged that the hospital and the attending physician failed to provide proper care and attention to her during labor and delivery, which failure resulted in injury to her and a spastic condition to the child.

Although the patient was in labor and the head of the child protruded, the ward nurse failed to call a doctor. As the patient was placed on the delivery table, the baby "shot out more than her own length on the delivery table. There was a thud, the baby hitting the table".

An award of $55,000. to the infant and $2,000. to her father was made against the hospital. It was the duty of the nurse, declared the court, to call an intern when she thought the patient's condition required the services of a physician. It was the duty of the intern to respond to that call, if possible; or for the nurse to call the private physician when true labor began. It was the further duty of the hospital, when the process of delivery was obviously imminent or had commenced, to endeavor to furnish an intern or other physician to act until the privately employed doctor could arrive.

(Garfield Memorial Hospital v. Marshall, 1 CCH Neg. Cases 2d, 1025—April 30, 1953—D.C.)

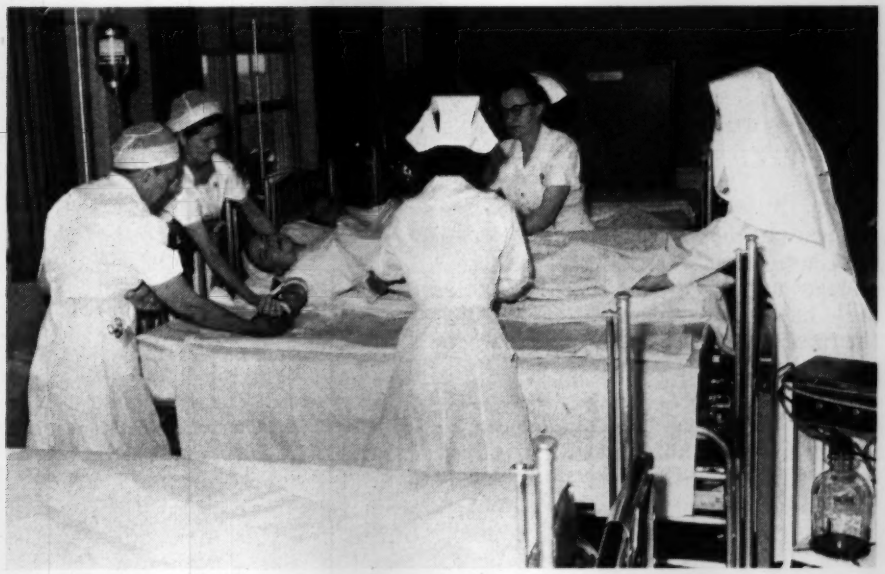
Notes and Case Reports

Surgical Recovery Room

Surgical Recovery Rooms differ from Anesthesia Recovery Rooms in that the patients remain in Surgical Recovery not only during the reaction from anesthesia, but from twelve to twenty-four hours. After this length of time the danger of immediate post-operative complications such as hemorrhage, delayed or secondary shock etc. is usually past. The patients are established on their post-operative regimes and orientated to them.

At St. Joseph's the Recovery Room is adjacent to the surgical unit, being separated only by large double doors. Two rooms, one on either side of the corridor, provide ten beds for men, ten beds for women. Segregation of the sexes is necessary since the patients remain long after they are orientated. The rooms are air conditioned. There is a wall oxygen outlet for each two beds, making oxygen easily available by either nasal catheter or mask. Oxygen tents are available for thoracic cases and for others on order of the surgeon. Suction machines, gastric suction apparatus, set-ups for irrigation and aspiration for urological patients, tracheotomy sets etc. are at hand.

Portion of Surgical Recovery showing Supervisor and chief Anesthetist in consultation in Nurses' station.



Transferring Patient from carriage to bed with intravenous fluids running.

In the work room are supplies of all of the usually ordered drugs: narcotics, sedatives, stimulants, antibiotics, parenteral fluids etc. as well as pharyngeal and naso-pharyngeal air ways and such items. Since the anesthesia room is not more than fifty feet away an endo-tracheal tray is not kept in Recovery.

All adults who have received anesthesia, either general or spinal, are admitted to Recovery. Those who have had minor surgery may remain only a few hours. Their beds may be used two or three times in a twenty four hour period. Those who have had major surgery will remain until the next morning. An occasional patient who is not in condition to be moved or who is critically ill and cannot afford special nurses, may remain a second day and night. Accident cases, such as brain or chest injuries or multiple compound fractures, may remain for two or three days, or until it is safe to move them. These patients usually have special nurses. Special nurses may attend any patient in Recovery but the usual procedure is to order nurses beginning the morning after the day of surgery for those who need or want them.

Children are not admitted to Recovery but are returned to Pediatric division.

One relative is permitted to see each patient as soon as they are admitted to Recovery and the immediate post operative procedures instituted. One relative may see them at two P.M. and one at seven P.M. for a few moments only.

Patients are transferred from surgery by Hausted carriage or on Davis Rollers. The anesthetist always accompanies the patient to Recovery, with some member

of the surgical staff; a nurse, an aid or an orderly. The anesthetist supervises and aids in placing the patient in bed in the proper position, reports on the anesthetic and surgical procedure and course, gives to the Recovery Room nurse the post-operative orders, checks the pulse and general condition after the moving, checks any fluids or blood that may have been transported with the patient and makes sure of a patent air way.

Post-operative orders, such as oxygen therapy, gastric suction, irrigation and aspiration, parenteral fluids, antibiotics etc. are instituted immediately the patient is admitted to Recovery. Levine tubes are passed by the anesthetist at the close of the anesthesia, if gastric suction has been ordered. Indwelling catheters are placed in surgery, if ordered. No time is then wasted in organizing the post-operative procedures. A "stir up regime" of deep breathing, leg exercises and turning is carried out every hour until the patient is well reacted and moving about voluntarily; then every two hours during the day and every three hours during the night. Blood pressure, pulse and general condition are checked at this time; oftener if indicated.

Transfer of patients to their rooms begins about five A.M. and by seven-thirty A.M. the beds are ready for new day's schedule.

The Surgical Recovery Room is staffed through the nursing office but has its own Supervisor who has no other duties or responsibilities. It is staffed on a twenty four hour basis except Sundays and holidays, when it is closed. The personnel for the three shifts, at present, consists of:

7 A.M. to 3 P.M.
2 R.N.s 1 L.V.N
3 student nurses orderly on call
3 P.M. to 11 P.M.
2 R.N.s 1 undergraduate
orderly on call
11 P.M. to 7 A.M.
1 R.N. 2 L.V.N.s
orderly on call.

The charge for Recovery is ten dollars (if the patient is not held over the twenty four hour period) plus medications used. The charge for their hospital room carries through but the room is available for a relative who may wish to remain near the patient but cannot stay in Recovery. The seeming extra expense is also counter-balanced by the saving of the expense for special nurses which would be far greater, if they were obtainable.

Our Surgical Recovery Room has been in operation on this basis for eight years, under the same Supervisor. On most days the twenty beds are all in use, some of them more than once. In addition to giving to all operative patients care that is equivalent to special nursing care, it has provided training for student nurses in post-anesthetic and post-operative care and refresher work for a number of nurses who have felt the need because of changing procedures, apparatus and drugs.

The Recovery Room staff and the Anesthesia staff have worked together very closely. A mutual appreciation has made very pleasant relations between the two groups. There is always an anesthetist quickly available for ad-

(Continued on page 71)

Abstracts

SALASSA, R. M.; BENNETT, W. A.; KEATING, F. R., JR., AND SPRAGUE, R. G.: Postoperative adrenal cortical insufficiency; occurrences in patients previously treated with cortisone. J.A.M.A. **152**:1509-1515 (Aug. 15) 1953.

"There is considerable evidence to support the view that the administration of adrenal cortical hormones may suppress adrenal function and induce adrenal atrophy. The extent, duration, and potential seriousness of this hazard in medical and surgical practice are, however, only now becoming apparent. A year ago, Fraser and co-workers reported a postoperative death that was regarded as the result of acute postoperative adrenal cortical insufficiency associated with atrophy and functional suppression of the adrenal cortex induced by previous treatment with cortisone. We wish to describe two additional examples of the same surgical complication, with further observations on the occurrence in man of adrenal atrophy induced by cortisone. . . .

"Case 1. A woman, 54 years of age, entered the Mayo Clinic on Oct. 31, 1951, because of rheumatoid arthritis and epigastric distress. . . . Beginning one year before admission, she was given 75 mg. of cortisone three times a week. After four months, the dose was increased to 100 mg. a day. She gained 20 lb. (9.1 Kg.), and noticed an increase in roundness in the face and a coarsening of features. For three or four weeks before admission she complained of epigastric distress that occurred between meals, and, because of this, the dose of cortisone was reduced to 75 mg. per day. . . . On Nov. 7, the patient vomited dark-brown material and suffered severe pain in the epigastrium. The blood pressure dropped, and she became cold and restless. Three transfusions totalling 2,000 cc. were given. The dose of cortisone during this period was 50 mg. per day by mouth. On Nov. 12, the dose was reduced to 37.5 mg. This dose was continued until 6 p.m., Dec. 5, when an additional 100 mg. of cortisone was given intramuscularly. On Dec. 6, a subtotal gastrectomy . . . was carried out. The patient remained semiconscious after the operation; signs of shock appeared; and she became anuric. Despite the intramuscular administration of 300 mg. of cortisone, the woman failed to respond to emergency measures and died 30 hours after operation. . . . The shock and anuria that followed operation in this patient did not seem commensurate with the uneventful operation and anesthesia that preceded it. The appearance of hypercortisonism and the changes observed in the adrenal and pituitary glands at necropsy constituted the only positive necropsy findings apart from pulmonary edema. They appeared to offer adequate evidence for the assumption that the final shock was due to acute adrenal cortical

insufficiency resulting from prior cortisone therapy.

"Case 2. A woman, 54 years of age, entered the Mayo Clinic on Aug. 18, 1952, for treatment of rheumatoid arthritis. . . . In 1949, the patient was treated with colloidal gold and in 1950 and 1951, she received four courses of cortisone or corticotrophin (ACTH) by injection; this was interspersed sporadically with cortisone given orally. During the preceding year, she received two injections of cortisone a week, and, on eight occasions, two injections per day for one week. During the vigorous treatment with cortisone, she noticed a rounding of her face. A year previously, a cystocele and a rectocele had been repaired without untoward reaction while the patient was receiving substantial doses of cortisone. . . . Treatment with cortisone was discontinued on the patient's admission. A total of 422 mg. of hydrocortisone was injected into the knees, occasions between Aug. 23 and Dec. 22. There was little untoward reaction to withdrawal of cortisone after the first two weeks. Signs of hypercortisonism persisted for about six weeks after withdrawal of the drug. On Nov. 3, urinary excretion of 17-ketosteroids was 1 mg. per 24 hours. On Jan. 7, 1953, a bilateral bunionectomy was performed. The patient withstood this procedure well, but 15 hours after operation irreversible shock developed. Despite treatment with 125 mg. of cortisone given intramuscularly and 100 mg. of hydrocortisone given intravenously 30 minutes before death, the woman died 16 hours after the operation. . . . The long interval without cortisone undoubtedly led to the false assumption that a mild surgical procedure could be undertaken without preoperative treatment. . . . Atrophy of the adrenals and pituitary constituted the only significant necropsy findings and provided anatomic support for the conclusion that the suppressive effect of prior cortisone therapy was the cause of the postoperative adrenal cortical crisis and death, even though treatment with cortisone had been discontinued four and a half months before. . . . .

"In order to investigate the occurrence of adrenal atrophy further, the weights of the adrenal glands of 46 adult patients who died of various conditions for which they had received cortisone were compared with the weights of the adrenal glands of 42 patients who died of similar conditions but who had never received cortisone. . . . While the average adrenal weight was significantly decreased in the group treated with cortisone, conspicuous adrenal atrophy occurred in only a small proportion of cases. . . . In nine patients who received cortisone for less than five days (four received it until the day of death), adrenal weights were always within the normal range and histological changes were lacking. The most consistent reduction in adrenal weight occurred in 10 patients who received cortisone for five or more days and until the day before death. The mean weights of the adrenals were significantly reduced in the groups of patients who were treated with cortisone for five or more days and in whom treatment with cortisone was discontinued within 20

days before death; in the groups of patients in whom treatment with the drug was discontinued for at least 21 days before death there was no significant reduction in the mean weights of the adrenals. The data obtained are too limited to permit further corelation with either dose or duration of therapy. It is important to note, however, that pronounced adrenal changes were observed on histological examination in a great majority of the patients who received cortisone, even though the combined weights of the adrenals were well within the normal range. . . .

"The changes in the adrenal cortex that seem to be characteristic of previous cortisone administration appear to be loss of lipid content of cells of both the zona glomerulosa and the zona fasciculata. Where this has occurred, an eosinophilic granular cytoplasm is seen. The cells shrink and cause decreased thickness of the cortex, but there is no apparent decrease in the number of cortical cells. Rather, they appear to be compressed, with an increase in the nuclear-cytoplasmic ratio. In adrenals examined after treatment with cortisone has been omitted for several weeks or months, evidences of recovery are seen. These consist of the appearance of more lipid material in the cells of the zona glomerulosa and the outer portions of the zone fasciculata. This material seems to replace the granules of the cytoplasm with vacuoles of varying size that, on frozen section, are filled with sudanophilic substance or lipid. This process proceeds in a patchy fashion, and in some areas there may be island-like projections throughout the thickness of the cortex. The zona reticularis loses its lipid last and often maintains a certain amount of lipid even in the severest cases in which it is depleted in the other zones. . . .

"From the evidence presented it seems reasonably clear (1) that cortisone is capable of producing atrophy of the adrenal cortex, (2) that this atrophy may be associated with impaired adrenal cortical function, (3) that both the atrophy and the accompanying functional impairment may outlast (perhaps occasionally for a long time) the withdrawal of cortisone, (4) that adrenal atrophy and impaired function may lead to acute adrenal cortical insufficiency in the face of sufficient stress, such as an operation, and (5) that these effects are due entirely to suppressed production of endogenous corticotrophin by the patient's pituitary. This implies, of course, that administration of corticotrophin may have the same hazard as the administration of cortisone. It has been suggested that the use of corticotrophin instead of, or together with, cortisone or hydrocortisone will eliminate the risk of impaired adrenal insufficiency. From what is known of the action of exogenous corticotrophin, this seems quite improbable. . . .

"The large number of conditions for which cortisone is employed and the large number of patients to whom it is given represent an important clinical problem that must be considered whenever any patient is to undergo surgical treatment. The clinical problem entails (1) the selection from among patients who have ever been treated with cortisone or corticotrophin those likely to have

sufficiently impaired pituitary and adrenal function to be liable to the development of adrenal cortical insufficiency as a result of surgical stress, (2) the prevention of adrenal cortical insufficiency in those patients whom the clinician considers susceptible, and (3) the treatment of postoperative adrenal cortical insufficiency when it appears in those instances in which clinical acumen was poor or prophylactic replacement therapy was withheld or inadequate. It is difficult, at present, to decide which patients are liable to adrenal cortical insufficiency as the result of previous therapy with adrenal or pituitary hormones. . . .

"How then is one to determine which few patients are liable to adrenal insufficiency and should receive prophylactic replacement therapy? If all patients are to be protected from all potential hazards, we may be obliged to act on the basis of what we do not know rather than on the basis of what we do know. We do not know the minimal or maximal interval that must elapse after withdrawal of cortisone before the suppressive effect may be considered to be certainly abolished. It would be most unwise to assume that this interval is short. On present evidence, it seems safest to suppose that any patient who has received cortisone in significant quantities within three to six months should receive prophylactic therapy. Patients with marked hypercortisonism (whether of natural origin or induced by medication) may be more liable to persistence of impaired pituitary and adrenal function than those who have merely had maintenance therapy. . . . We feel that any patient who has had severe hypercortisonism within one to one and a half years before an operation should perhaps be treated as though he were liable to acute adrenal insufficiency. We do not know the shortest period of treatment that will produce adrenal suppression. . . . It is difficult to say what reliance should be placed on tests of adrenal cortical function. . . .

"The only entirely reassuring measure of the ability of the adrenal cortex to function safely would be its response to a stress comparable to that represented by the proposed surgical procedure. Since this is not possible at present, every patient who has been exposed to cortisone therapy in the terms that have been described should be regarded during and after a surgical procedure as potentially liable to the development of postoperative adrenal cortical insufficiency. . . . In those instances in which the clinician regards postoperative adrenal cortical insufficiency a serious hazard, preoperative or prophylactic treatment may be given. . . .

The following regimen may be proposed: 200 mg. of cortisone are given intramuscularly 48 hours, 24 hours, and 1 or 2 hours before operation. Cortisone given orally before operation may be of no advantage since its effects are of short duration and hence no steroid depot, such as is desired, is thereby created. If preoperative cortisone therapy is given, the dose of cortisone is gradually reduced after the operation. The rate of reduction of the dose and the duration of therapy after operation are determined by the condition of the patient. Usually

treatment with cortisone is discontinued in three or four days. There seems to be no reason to fear untoward effects of such doses provided they are not given for long periods of time.

"We believe that all patients who give a history of prior therapy with cortisone, regardless of whether they have been considered suitable subjects for preoperative or prophylactic treatment, should be followed postoperatively as though the surgeon expected to encounter adrenal cortical insufficiency. They should be closely observed, particularly during the first 24 hours after operation. Temperature, pulse, and blood pressure should probably be recorded at hourly intervals during this period. Certain special precautions may, in some instances, enable patients to avoid difficulty. They should not be made to fast for an excessive period of time. If possible, the operation should be performed early in the morning rather than late in the day. Furthermore, patients should not be given excessive quantities of 5% solution of glucose and distilled water. The postoperative management of the water and electrolyte balance in patients with real or potential adrenal insufficiency is at best a difficult matter, but there seems to be no doubt that such patients do not tolerate excessive quantities of water. For this reason, glucose without sodium chloride solution should probably not be given. Also, such patients for reasons that are not understood, are sometimes adversely affected by morphine or its derivatives. Morphine or related substances, if given at all, should be used in small quantities; it would perhaps be best to use alternative types of narcotics.

"If, in spite of all efforts, adrenal cortical insufficiency does occur, it appears that it is most likely to happen during the first 24 hours after operation. It is perhaps most likely to appear as a sudden circulatory collapse, fall in blood pressure, rise in pulse rate, and, perhaps, elevation of temperature. The patient may become unconscious rather quickly. Any of these symptoms should be regarded as indicating a serious emergency. The administration of an isotonic solution of sodium chloride or 5% glucose in an isotonic solution of sodium chloride should be started at once. If solutions of cortisone or hydrocortisone for intravenous use are available, they may be lifesaving in such circumstances and should be given promptly. If solutions of steroids for intravenous use are not available, large quantities of aqueous adrenal extract should be administered both intramuscularly and intravenously (the latter by adding it to the sodium chloride solution administered intravenously). Four milligrams of norepinephrine added to the sodium chloride solution appears to aid in controlling circulatory collapse and extreme hypotension. Although it may have no immediate effect, 200 mg. of cortisone may be given intramuscularly, since it will ultimately become effective if the patient can be maintained during the first 24 hours by more rapidly acting preparations. If a patient is conscious and not nauseated or vomiting, 100 to 200 mg. of cortisone may be given orally."

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Book Reviews

Principles of Thoracic Anesthesia. Past and Present. By William W. Mushin, M.A. (Oxon.), M.B., B.S. (Lond.), M.R.C.S., F.F. A.R.C.S., D.A., Director of Anaesthetics, Welsh National School of Medicine, Consultant Anaesthetist, United Cardiff Hospitals, Consultant Advisor in Anaesthetics, Welsh Regional Hospital Board, Formerly First Assistant, Nuffield Department of Anaesthetics, University of Oxford, and L. Rendell-Baker, M.B., B.S. (Lond.), M.R.C.S., D.A., Consultant Anaesthetist, United Cardiff Hospitals and Welsh Regional Hospital Board; First Assistant, Department of Anaesthetics, Cardiff. Cloth. 172 pages, 217 illustrations, Springfield: Charles C Thomas, 1953.

The authors have paid tribute to the many surgeons and anesthetists who have built the basic foundations for the present principles of anesthesia for thoracic surgery. The problem of the pneumothorax is first discussed. Following this a thorough discussion of the historical background for present practice is presented. Methods of anesthesia for thoracic surgery in use at this time constitutes the third section of the book. No attempt has been made to include details of technic since the authors believe that present day technics are as likely to change as those of years ago. Much emphasis has been placed on the mechanics and the devices utilized in the practice of anesthesia for thoracic surgery. Pictures are used extensively; in some instances identical pictures are reproduced in a second section of the book. Persons interested in anesthesia for thoracic procedures will find this an extensive discussion of some of the problems confronted and their solutions. In the appendix pictures of many of the physicians who contributed to the development of this phase of anesthesia are accompanied by biographical notes. Each chapter of the book is followed by a list of references.

Resuscitation of the Newborn. By Joseph D. Russ, M.D., F.A.A.P., Assistant Professor of Pediatrics, Tulane University School of Medicine, Senior Pediatrician Touro Infirmary, New Orleans, Louisiana. Cloth. 55 pages. 7 illustrations. Springfield: Charles C Thomas, 1953. $2.50.

This monograph is another in the American Lecture Series. Following the pattern used in this series the author has brought together in concise and orderly manner current information supplemented by his own work to present a detailed picture of the problem of asphyxia of the newborn. The volume is concluded with a plea for education of all persons involved in the care of the newborn in order that this major cause of neonatal complications may be reduced. 139 references are included. This book would seem to be a "must" for all anesthetists.

Classified Advertisements

ANESTHETIST: 110-bed hospital. Work light. Live in or out. Salary $400 plus bonus and percentage on work. Mrs. Probandt, Superintendent, Belmont Community Hospital, 4058 W. Melrose Street, Chicago, Illinois; Pensacola 6-7000.

NURSE ANESTHETIST, OBSTETRICAL: 373 bed general hospital; expansion to 573 completed within year. A.C.S., A.M.A., and A.H.A. approved. New facilities; pleasant working conditions; forty hour week; over 3,000 deliveries in 1952. Minimum salary $335 monthly or higher depending on qualifications. For further detail apply: R. H. Harter, Personnel Director, Aultman Hospital, Canton, Ohio.

NURSE ANESTHETIST: 145 bed general hospital in S.W. Pennsylvania. Two anesthetists now employed. Hospital expanding. Excellent working conditions. Salary $400 and comfortable living accommodations. Apply: Administrator, Greene County Memorial Hospital, Waynesburg, Pa.

NURSE ANESTHETIST WANTED to work in anesthesia department, general hospital with 155 beds. Good working conditions; four nurse anesthetists on staff at present time. Salary $400 base, plus overtime. Contact: Miss Ora Hartley, Head Anesthetist, or the Personnel Office at Beyer Memorial Hospital, Ypsilanti, Mich.

NURSE ANESTHETIST: 200 bed general hospital. Salary open, plus benefits. Staff of 3 anesthesiologists and 2 nurse anesthetists. Apply Chief of Anesthesiology, French Hospital, San Francisco, California.

NURSE ANESTHETIST WANTED To work in anesthesia department general hospital, 289 beds. Liberal salary, 40 hours week, good personnel policies. Four anesthetists on staff at present time. Write or call Holston Valley Community Hospital, Kingsport, Tenn.

NURSE ANESTHETIST - For approved general hospital. Must be able to administer latest types anesthetic agents. Salary $310 to $360 per month plus full maintenance. Annual vacation and sick leave. Retirement benefits if desired. Apply Administrator, Robinson Memorial Hospital, Ravenna, Ohio.

WANTED: nurse anesthetist 65 bed modern hospital 5 1/2 day week salary $500 including board and laundry. Contact Hazel E. Carbaugh, Administrator, Alleghany Memorial Hospital, Covington, Va.

WANTED: Nurse anesthetist for 86 bed hospital. Salary open. No O. B. calls. Liberal employee benefits. Apply Superintendent, Group Health Hospital, 201 Sixteenth Avenue North, Seattle 2, Washington.

ANESTHETISTS: A.A.N.A. members. 250 bed general hospital; salary open; automatic increases; laundry provided; forty hour week; no obstetrics; liberal vacation and personnel policy; Social Security. Sutter Hospital, Sacramento, Calif.

WANTED: Two nurse anesthetists for 240 bed hospital. Salary open. Partial maintenance provided. Apply: Administrator, Charleston General Hospital, Charleston, W. Va.

REGISTERED NURSE ANESTHETISTS. Permanent and vacation relief positions open for surgery and obstetrical departments. Paid overtime. Extra pay for night duty. Automatic pay increases. Living accommodations available. Apply: Chief nurse anesthetist, Harper Hospital, Detroit 1, Mich.

WANTED: Nurse anesthetists to increase personnel. For details write Robert A. Hingson, M. D., Director, Department of Anesthesia, University Hospitals of Cleveland, 2065 Adelbert Road, Cleveland 6, O.

NURSE ANESTHETIST, female: 225 bed hospital, resort city in southern Connecticut. L beral salary and many benefits. Apply: Personnel Office, Lawrence and Memor al Associated Hospitals, New London, Conn.

ANESTHETIST - Registered Nurse, with two or more years' experience, for 200 bed hospital on Florida's Gulf Coast. Medical Anesthesiologist in charge of department. Good salary, living quarters, yearly vacation and sick leave. Write Box M-24, The Journal, A.A.N.A. 116 S. Michigan Avenue, Chicago 3, Ill.

ANESTHETISTS WANTED: Busy suburban hospital near Chicago. New nurses' residence. (Apartments available for married anesthetists.) Starting salary $330 per month and complete maintenance which is equivalent to $450. After sixty days $345 per month. Vacation, sick leave, Blue Cross insurance and free life insurance. MacNeal Memorial Hospital, Berwyn, Illinois.

NURSE ANESTHETISTS for general non-profit 316 bed hospital. Salary open. Complete maintenance. Excellent personnel policies, including vacation, sick leave, and hospitalization insurance. Eastern Virginia. Write Director of Anesthesia, Norfolk General Hospital, Norfolk, Va.

WANTED — ANESTHETISTS. 450 bed teaching hospital. Department directed by medical anesthesiologist. Southern city with cultural advantages. $400 per month with full maintenance; periodic increases in salary; liberal vacation and sick leave; hospitalization and pension plan benefits. Apply: C. A. Robb, Superintendent, Roper Hospital, Charleston, S. C.

WANTED: 2 Nurse Anesthetists to increase staff. Working conditions excellent. Apply Chief, Anesthesia Department, The Mercer Hospital, Trenton, N. J.

ANESTHETIST Nurse for new 50 bed hospital, fully approved. Good working conditions. Salary $450. and other benefits. Apply DELNOR Hospital, St. Charles, Ill.

REGISTERED NURSE ANESTHETIST: Modern, suburban, 230 bed hospital 30 minutes from Philadelphia, Pa Department directed by medical anesthesiologist. $365 per month plus laundry. May live in, very reasonable, or out. Surgery schedule usually completed by noon. On call five weekdays, one week end per month and have each following day off. Week ends off: two at Friday, one Saturday noon. Month vacation. Wilmington General Hospital, Wilmington, Del.

WANTED: Nurse anesthetists for 500 bed University teaching hospital; starting salary $383 per month. Stated increases; vacation and holiday leave; cumulative sick leave. Apply: Anesthesiologist in Charge, University of Virginia Hospital, Charlottesville, Va.

NURSE ANESTHETISTS (TWO): A.A.N.A. members. 300 bed hospital; medical director in charge of department; hospital located in city of 150,000 people. Mercy Hospital, 2000 East Fifth Street, Charlotte 4, N. C.

NURSE ANESTHETIST: To increase present staff of 10 nurse anesthetists. A.A.N.A. membership or eligibility for membership required. Starting salary $375 per month with yearly increase, plus full maintenance or cash allowance for maintenance, as desired. Private room with bath and telephone in new women's residence. Social Security and private pension plan. Excellent working conditions; surgical and delivery suites air conditioned. 44 hour week. Apply: Marshall Kerry, M.D., Chief Anesthesia, The Reading Hospital, Reading, Pa.

NURSE ANESTHETISTS—two for 200 bed general hospital, Anesthesiologist in charge. Salary $450-$500. Paid vacation and holidays. Extra compensation plan. Baptist Hospital; Birmingham, Ala.

NURSE ANESTHETIST: For 200 bed general hospital. 40 hr. week, with attractive salary. Nurse anesthetist in charge. Maintenance available. Contact Catherine Little at Fairview Park Hospital, Cleveland, O.

NURSE ANESTHETIST: Approved hospital near Detroit. $450 per month. Overtime after forty hours per week. Living quarters available. Wyandotte General Hospital, Wyandotte, Mich.

WANTED: three nurse anesthetists, 100-bed hospital, to give surgical and obstetrical coverage, 40-hour week except every third week-end—salary, $375. Apply, Executive Director, Kent General Hospital, Dover, Del.

ANESTHETISTS: $4764 to $5004. For 40 hour week. Positions in Wayne County General Hospital and Infirmary, Eloise, Michigan, 16 miles west of downtown Detroit. Paid vacations and sick leave. Time and a half for overtime. Extra pay for standby service. Contact: Civil Service Commission, County of Wayne, 2200 Cadillac Tower, Detroit 26, Michigan.

INHALATION THERAPY

(Continued from page 25)

treatment of diseases and conditions of the lungs accompanied or complicated by the presence of excessive or thickened broncho pulmonary secretions (such as bronchial asthma, acute and chronic purulent bronchitis, bronchietasis, emphysema, atelectasis, pneumonitis). Such secretions may accompany general anesthesia, thoracic surgery, tracheotomy, aspiration of materials such as foreign bodies, gastric contents or amniotic fluids.

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PITTSBURGH: General hospital, 600 Beds, needs 2 or 3 additional nurse anesthetists - Department now consists of 8 nurses and one anesthesiologist as director - No obstetrics - Four weeks paid vacation per year - Forty hour week - Overtime paid on hourly basis. Apply: Dr. B. Gillespie, M.D. Dept. of Anesthesia, West Penn Hospital, Pittsburgh 24, Pa.

NURSE ANESTHETIST, 45-bed general hospital, new surgery, modern anesthesia equipment, no O. B. Very attractive salary. Unusual opportunity to increase present earning power. Position open April 1, 1954. Contact Administrator, Victory Memorial Hospital, Stanley, Wis.

WANTED: Nurse anesthetist. Starting salary $300 plus complete maintenance and $10 each night on call. On call duty four to six nights monthly. Eight nurse anesthetists on staff. Three weeks' vacation and twelve sick leave days per year. Automatic annual pay increase and bonus. Apply: Mrs. F. Stowe Burwell, Charlotte Memorial Hospital, Charlotte, N. C.

RECOVERY ROOM

(Continued from page 60)

vice or assistance; for emergency intubations, suctioning or help with difficult veni-punctures. The anesthetists can, and do, check the patients in Recovery frequently; much more frequently than we could were they scattered throughout the hospital. We believe that our low incidence of post-anesthetic and post-operative complications is due, in large part, to the constant, expert care provided by the Surgical Recovery Room during the first twelve to twenty four hours.

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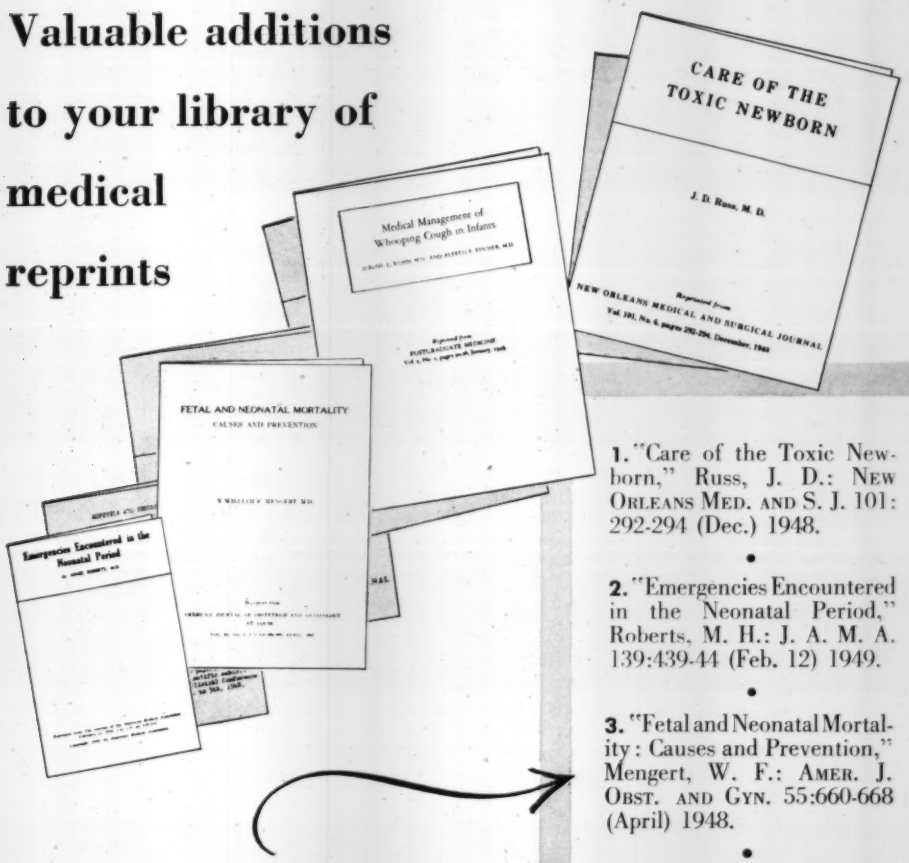

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2. "Emergencies Encountered in the Neonatal Period," Roberts, M. H.: J. A. M. A. 139:439-44 (Feb. 12) 1949.

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4. "Medical Management of Whooping Cough in Infants," Kohn, J.L. and Fischer, A.E.: POSTGRAD. MED. 5:20-26 (Jan.) 1949.

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